THE ASSISTANT SECRETARY OF DEFENSE

WASHINGTON, D. C. 20301-1200

HEALTH AFFAIRS

MEMORANDUM FOR SURGEON GENERAL OF THE ARMY
SURGEON GENERAL OF THE NAVY
SURGEON GENERAL OF THE AIR FORCE

SUBJECT: Clinical Information on Leishmania

REFERENCE: DoD Message dated 8 November 1991, subject line Deferral of Military Donors

Per our discussions, the attached information paper is provided for immediate dissemination to all appropriate personnel to include all clinicians.

I request that you disseminate this information as rapidly as possible to allay unwarranted anxiety on the part of our patients and clinicians.

Enrique Mendez, Jr., M.D.

Attachement:
As stated

#561

Dear Doctor:

This is to inform you of a viscerotropic form of leishmaniasis due to the parasite Leishmania tropica among military personnel who deployed to Southwest Asia (SWA) during the Gulf War. To date, seven cases have been diagnosed at Walter Reed Army Medical Center. Normally, an infection with Leishmania tropica results in cutaneous lesions only; however, all of these cases were free of cutaneous lesions and, in each case, the parasite was recovered from the bone marrow. These new cases are distinct from the traditional cutaneous form of leishmaniasis of which 15 Gulf related cases have been diagnosed and treated at Walter Reed Army Medical Center.

Epidemiologic risk factors for this viscerotropic form are not well defined at this time. These seven soldiers were members of several different Army units widely scattered throughout the SWA theater of operations in both field and urban settings. Navy, Marine, Air Force and civilian personnel who were stationed within the theater of operations are also considered at risk of exposure.

The natural history of this viscerotropic form of L. tropica is not known. The fact that it has not been clinically apparent in the many travelers to and inhabitants of that region suggests that infections are rare, and/or largely subclinical. Based on the current cases, the clinical appearance is much less severe than that seen in classical visceral leishmaniasis (Kala Azar) caused by L. donovani. As with other parasitic and infectious diseases in the immunosuppressed patient, L. tropica has the potential for causing serious illness.

The clinical spectrum for these cases was variable and nonspecific. Four of the six symptomatic cases had an acute syndrome which included a high fever with rigors and malaise, accompanied by mild anemia and low grade elevation of liver enzymes (AST and ALT). Two cases had a subacute onset, presenting with gastrointestinal complaints which included watery, fecal-leucocyte-negative diarrhea (of small volumes), nausea, and non-focal abdominal pain that evolved over time to left upper quadrant pain with hepatosplenomegaly. Headaches and chronic irritating cough were also seen in some cases. One of the seven cases was completely asymptomatic and diagnosed on the basis of epidemiologic follow-up of an index case.

The incubation period is difficult to accurately measure. However, in these cases, the onset of symptoms varied from weeks to months after leaving SWA.

A serum Indirect Immunofluorescent Antibody (IFA) test is available at Walter Reed Army Institute of Research (WRAIR) through the Walter Reed Army Medical Center, however, there is no commercially available serologic test currently available in the United States to confirm infection. With this test, in patients

with suggestive signs and symptoms of this leishmania infection, a serum IFA equal to or greater than 1:32 suggests infection. Diagnosis can be confirmed by identifying the organism in tissue and culturing a bone marrow aspirate. Smears of the aspirate were negative by Giemsa stain, but the organism was detected at WRAIR using anti-leishmania monoclonal antibodies tagged with fluorescein.

The treatment of choice is sodium stibogluconate (Pentostam), an investigational new drug (IND) produced by Wellcome Trust of Great Britain. It is available under protocol to military physicians at Walter Reed Army Medical Center through Walter Reed Army Institute of Research, Washington, DC and to civilian physicians from the Centers for Disease Control (CDC), Atlanta, Georgia. Five of these seven cases were treated with sodium stibogluconate. In two of these five cases, treatment was discontinued early because of thrombocytopenia. Following treatment, all five patients recovered, returned to duty and are being followed. A sixth case was asymptomatic, therefore, not treated with sodium stibogluconate and is being followed closely. The seventh and most recent case is still under care at Walter Reed Army Medical Center.

The military Medical Departments are actively increasing their case finding efforts by heightening awareness of both military and civilian physicians to this clinical situation. Since many of our military personnel who deploy to the Gulf were reservist who, if ill, would visit their personal civilian physician in the private sector, the CDC and FDA are participating in this effort.

Transmission of the organism is by the bite of a sandfly. Man is an incidental host. Person-to-person transmission of this form has not been reported.

There have been five cases reported in the world literature of visceral leishmaniasis (L. donovani) transmitted through blood transfusions. No case of transfusion-associated transmission has been reported with L. tropica, but a theoretical risk exists. Therefore, as a conservative safety precaution due to this theoretical risk and the absence of a simple screening test, the Assistant Secretary of Defense (Health Affairs) has directed the military services to defer, until further notice, all personnel form blood donation who were stationed in Saudi Arabia, Kuwait, Iraq, Bahrain, Qatar, United Arab Emirates, Oman and Yemen from 1 August 1990 and thereafter.

Blood products already collected from Gulf War returnees currently in the inventory are not being withdrawn because of the very low risk of contamination balanced against the risk of a sudden shortfall of critical blood supplies. Under current procedures, donors who were symptomatic or blood units with an elevated ALT level would have already been excluded. None of the identified cases have donated blood and all patients with any form of leishmaniasis are permanently deferred from future donations.

A blood donation history will be taken from any future case and, if positive, a complete lookback for that case will be conducted.

The potential threat of exotic pathogens in Southwest Asia was reviewed by military medical planners prior to the Gulf War and is outlined by the New England Journal of Medicine article published on 21 March 1991 by Gasser, et al, and Supplement Number 1 to Reviews of Infectious Disease, January 1991 by Oldfield, et al. These are excellent references for practicing physicians.

Any physician who suspects leishmaniasis involving Department of Defense service personnel should contact the appropriate service Infectious Disease Consultant.

U.S. Air Force: Gregory Melcher, MD
MAJ, USAF
Wilford Hall Medical Center
Lackland Air Force Base
San Antonio, TX 78236
COM. TEL: (512) 670-7444
DSN: 554-7444
FAX: (512) 675-0173

U.S. Navy/Marines: Edward Oldfield, MD
CAPT, USN
Naval Hospital
San Diego, CA 92134
COM. TEL: (619) 233-2215
DSN: 987-2215
FAX: (619) 532-7484

U.S. Army: Charles N. Oster, MD or Alan Magill, MD
COL, USA MAJ, USA

Infectious Disease Service
Walter Reed Army Medical Center
Washington, DC 20307
COM. TEL:(202) 576-0585/0586
DSN: 291-0585/0586
FAX: (202) 576-2478

We recommend that, for other individuals, the CDC be contacted.

Centers for Disease Control
Atlanta, Georgia 30333
Drug Request: (404) 639-3670 (Monday-Friday)
or
(404) 639-2888 (Evening, Weekend, Holiday)

SIGNATURE

DEPARTMENT OF DEFENSE
OPERATIONS DESERT SHIELD/DESERT STORM
HEALTH EFFECTS SURVEILLANCE PROGRAMS

Since the earliest stages of the deployment of our troops to the Persian Gulf region during Operation Desert Shield, the Department of Defense has taken measures to monitor personnel for potential adverse health effects. During Operation Desert Shield, a surveillance system for the epidemiological monitoring of the occurrence rates of diseases and non-battle related injuries was in place. This monitoring system identified that the rates of illnesses and non-battle injuries were lower during both Operations Desert Shield and Desert Storm than in any previous deployment of U.S. Forces. In addition, this system allowed us to identify that there were no acute health effects from exposure to the smoke from the oil well fires in Kuwait.

As a part of the immediate actions taken to determine the acute health effects being experienced by individuals exposed to smoke from the oil well fires, the U.S. Navy conducted an epidemiological study of 2700 Marines in Theater to see if sick call rates correlated to potentially greater exposure. This study used self-reported questionnaires to identify each individual's potential level of exposure and any medical symptoms they were experiencing. The initial anaylsis indicated slightly increased levels of respiratory symptoms for Marines who were closer to the fire. However, there was no evidence of any serious, acute health effects.

In April, 1991, the Department established a Tri-Service working group on the Health Effects of the Kuwaiti Oil Well Fire smoke. This group is composed of personnel with expertise in occupational medicine, preventive medicine, environmental health, and industrial hygiene. In addition to the working group members from DoD, the Department has called upon individuals from the Department of Veterans Affairs, the Centers for Disease Control, the Environmental Protection Agency, the National Oceanic and Atmospheric Administration, the Armed Forces Institute of Pathology, and the Armed Forces Epidemiology Board.

The purpose of this working group is to develop and implement programs to identify adverse health effects which troops who served in the Persian Gulf may potentially experience and to recommend appropriate action to be taken to protect the health of our troops. Two major initiatives are currently underway to accomplish these goals; 1) the Kuwait Oil Fire Health Risk Assessment for DoD Troops, and 2) the Kuwait Oil Fire Biologic Surveillance Initiative. The interpretation of these studies is interdependent and the final report of their findings is expected to be completed in December, 1992.

The Health Risk Assessment is a study conducted in Kuwait and Saudi Arabia to determine the levels of contaminants that existed in the Region during the time the oil well fires were burning. In addition, this study will develop estimates of the levels of exposure individuals may have experienced while deployed in the Region. The protocol used in this survey is a multidisciplinary, matrixed plan based upon the Environmental Protection Agency's risk assessment guidance for Superfund sites.

The Biologic Surveillance Initiative is a prospective study of the health effects of deployment in the Persian Gulf Region being conducted on 4700 Soldiers of the 11th Armored Cavalry Regiment. The data for this study were collected during the period June 1 through October 14, 1991. These data include answers to health questionnaires, pulmonary function tests, the use of medical treatment facilities, personal diaries, and various analyses of biological specimens from a sample of individuals. This study is being conducted in collaboration with investigators from the Occupational Safety and Health Administration, the National Cancer Institute, the Center for Disease Control, and the Johns Hopkins Medical Institutions.

While these scientific activities are being conducted to delineate the potential health risks, action has also been taken to assure that individuals who may potentially be at risk for adverse health effects can be identified in the future. In November, 1991, the Department began working with the Department of Veterans Affairs to establish a registry of the individuals who served in the Persian Gulf Region. This registry will consist of a data base of all daily ground locations of military combat units that operated in the theater during Operations Desert Shield/Desert Storm. In addition, the Military Departments, the Joint Staff, and all other offices with relevant information have been directed to preserve all Persian Gulf records that contain information about troop rosters and unit grid coordinate locations.

In addition to these epidemiological studies, the Department has investigated four reports of disease clusters among individuals who served in the Persian Gulf. In November, 1991, seven unusual cases of infection with the parasite, Leishmania tropica, were identified. These cases, as well as about 15 cases of typical L. tropica infection, have been successfully treated at Walter Reed Army Medical Center. Also, a major research effort has been launched to develop tests to assist in the early diagnosis of infection with this parasite. Until the magnitude of the problem with this infection is confirmed, Service members who deployed have been barred from

donating blood as a precaution against contamination of the nation's blood supply. Each of the Services have disseminated information to all DoD health care providers, information about the signs, symptoms, and treatment of individuals who are infected with this parasite. The Reserves, the National Guard, the Centers for Disease Control, the State and Territorial Epidemiologists, and the State and Territorial Public Health Laboratory Directors have also been provided this information, an advisory about blood donations from Service members who served in the Persian Gulf, and procedures for referring individuals who are suspected of having this disease to appropriate sources of care.

The second reported disease cluster related to the perception of increased occurrences of miscarriages among U.S. Army personnel returning from Kuwait and Saudi Arabia and their families. The Office of the Surgeon General of the Army evaluated the incidence of spontaneous abortions at several of the Army's community hospitals on posts from which soldiers were deployed. Data obtained for the periods May through August of 1991 were compared with similar data from 1990. In all locations, the miscarriage rates were considerably lower than the national average which approximates 15 percent. Additionally, no difference was found in the rates for 1991 compared to 1990.

The third potential disease cluster relates to the incidence of post-traumatic stress disorder (PTSD) among personnel participating in Operation Desert Storm. As outlined in the Department's report to Congress last year, which will be updated this month, estimates based on our experience to date suggest that the maximum expected incidence of clinical PTSD among troops who served in the Persian Gulf, based on the total in theater force, would be about 100 individuals. All individuals diagnosed with post-traumatic stress disorder have received treatment. Based on the expectation that there will not be a sharp increase in PTSD cases, the present mental health resources will be sufficient to provide the necessary rehabilitative services. The Department will continue to monitor this potential problem closely.

The fourth reported disease cluster occurred among 120 reservists assigned to the 123rd ARCOM. These individuals complained of a variety of health related symptoms including chronic fatigue, joint/muscle aches, hair loss, aching teeth, bleeding gums, and thick saliva. A series of medical evaluations were conducted during March and April, 1992, including an assessment by an Epidemiologic Consultant Service (EPICON) team. The assessments concluded that most of the symptoms were due to unrelated medical, dermatological,

orthopedic, and dental problems. No evidence of a common toxic, environmental, vaccine-related, or microwave exposure causation was identified. Nonetheless, we will continue with follow-up evaluations.

The Department of Defense is aggressively evaluating each reported disease cluster and assuring that individuals receive necessary treatment for identified medical conditions. To date, there is no objective evidence to suggest any common causation for the symptoms/conditions that have been observed. This process of disease cluster surveillance will continue until it is clear that no outbreak of illness, caused by exposures during service in Kuwait and Saudi Arabia, will occur among individuals deployed to the Persian Gulf. In addition, the Department is currently designing an epidemiologic comparison study to be conducted in 1994 as the first of a series of recurrent studies to investigate the potential occurrence of adverse health effects resulting from service during Operations Desert Shield and Desert Storm.

DEPARTMENT OF THE ARMY
OFFICE OF THE ASSISTANT SECRETARY
WASHINGTON, DC 20310-0111

May 14, 1992

REPLY TO ATTENTION OF

MEMORANDUM THRU DIRECTOR OF THE ARMY STAFF F. DAVID GOLEMAN, LTC, GS, ADAS
21 MAY 1992

FOR THE DEPUTY CHIEF OF STAFF FOR PERSONNEL
THE SURGEON GENERAL

SUBJECT: Change in Policy for a Particular Disease Category

Particular details have come to my attention concerning soldiers of the Reserve Components who served in Southwest Asia and subsequently contracted Leishmaniasis. I have learned that this disease has a considerable and varied latency, or incubation, period between the time of exposure and development of symptoms. I also understand that if these soldiers had developed symptoms prior to their release from active duty, they would have qualified under our regulations to remain on active duty for treatment.

Although these soldiers are currently having their medical needs met, I do not like the perception that they are being penalized for not developing symptoms prior to REFRAD, when in fact this was through no fault of theirs and in fact the disease was contracted during their deployment in service to their country.

Please implement immediately the offer to each Reserve Component soldier diagnosed and being treated for Leishmaniasis the option to be returned to active duty for the duration of their acute phase treatment. The return should start the date the soldier is admitted to Walter Reed Army Medical Center for their evaluation for treatment. Also insure that each one has been placed on Incapacitation Pay effective from the time symptoms developed or diagnosis was made (whichever date resulted in incapacitation) through their return to active duty, or completion of acute phase treatment if return to active duty is declined.

9242776 Z

This is an exception to our recall to active duty for medical treatment policy for this specific disease entity only and should not effect any prior or future decisions concerning any other disease or injury.

G. Kim Wincup
Assistant Secretary of the Army
(Manpower and Reserve Affairs)

OPERATIONS
SUPPORT DIRECTORATE

ROUTINE ZYUW RHCGAAA4439 3171515
R 1312OOZ NOV 91
FM CDRFORSCOM FT MCPHERSON GA//FCMD-PM//
TO AIG 9879 AIG 9169
R 1218532 NOV 91
FM DA WASH DC//HODA//SGPS-CP-B//
TO CDR USAHSC FT SAM HOUSTON TX// CDR 7TH MEDCOM HEIDELBERG GE//
CDR18THMEDCOM SEOUL KOR // AIG 7446//

UNCLAS SECTION 01 OF 02
SUBJECT: LEISHMANIASIS IDENTIFIED IN SOLDIERS RETURNING FROM SOUTHWEST ASIA (SWA)

1. THIS IS TO INFORM YOU OF A VISCEROTROPIC FORM OF LEISHMANIASIS DUE TO THE PARASITE LEISHMANIA TROPICA AMONG MILITARY PERSONNEL WHO DEPLOYED TO SOUTHWEST ASIA (SWA) DURING THE GULF WAR. TO DATE, SEVEN CASES HAVE BEEN DIAGNOSED AT WALTER REED ARMY MEDICAL CENTER. NORMALLY, AN INFECTION WITH LEISHMANIA TROPICA RESULTS IN CUTANEOUS LESIONS ONLY; HOWEVER, ALL OF THESE CASES WERE FREE OF CUTANEOUS LESIONS AND, IN EACH CASE, THE PARASITE WAS RECOVERED FROM THE BONE MARROW. THESE NEW CASES ARE DISTINCT FROM THE TRADITIONAL CUTANEOUS FORM OF LEISHMANIASIS OF WHICH 15 GULF RELATED CASES HAVE BEEN DIAGNOSED AND TREATED AT WALTER REED ARMY MEDICAL CENTER.

2. EPIDEMIOLOGIC RISK FACTORS FOR THIS VISCEROTROPIC FORM ARE NOT WELL DEFINED AT THIS TIME. THESE SEVEN SOLDIERS WERE MEMBERS OF SEVERAL DIFFERENT ARMY UNITS WIDELY SCATTERED THROUGHOUT THE SWA THEATER OF OPERATIONS IN BOTH FIELD AND URBAN SETTINGS. NAVY MARINE, AIR FORCE AND CIVILIAN PERSONNEL WHO WERE STATIONED WITHIN THE THEATER OF OPERATIONS ARE ALSO CONSIDERED AT RISK OF EXPOSURE.

3. THE NATURAL HISTORY OF THIS VISCEROTROPIC FORM OF L. TROPICA IS NOT KNOWN. THE FACT THAT THIS HAS NOT BEEN CLINICALLY APPARENT IN THE MANY TRAVELERS TO AND INHABITANTS OF THAT REGION SUGGESTS THAT INFECTIONS ARE RARE, AND/OR LARGELY SUBCLINICAL. BASED ON THE CURRENT CASES, THE CLINICAL APPEARANCE IS MUCH LESS SEVERE THAN THAT SEEN IN CLASSICAL VISCERAL LEISHMANIASIS (KALA AZAR) CAUSED BY L. DONOVANI. AS WITH OTHER PARASITIC AND INFECTIOUS DISEASES IN THE IMMUNOSUPPRESSED PATIENT, L. TROPICA HAS THE POTENTIAL FOR CAUSING SERIOUS ILLNESS.

4. THE CLINICAL SPECTRUM FOR THESE CASES WAS VARIABLE AND NONSPECIFIC. FOUR OF THE SIX SYMPTOMATIC CASES HAD AN ACUTE SYNDROME WHICH INCLUDED A HIGH FEVER WITH RIGORS AND MALAISE, ACCOMPANIED BY MILD ANEMIA AND LOW GRADE ELEVATION OF LIVER ENZYMES (AST AND ALT). TWO CASES HAD A SUBACUTE ONSET, PRESENTING WITH GASTROINTESTINAL COMPLAINTS WHICH INCLUDED WATERY, FECAL-LEUCOCYTE-NEGATIVE DIARRHEA (OF SMALL VOLUMES), NAUSEA, AND NON-FOCAL ABDOMINAL PAIN THAT EVOLVED OVER TIME TO LEFT UPPER QUADRANT PAIN WITH HEPATOSPLENOMEGALY. HEADACHES AND CHRONIC IRRITATING COUGH WERE ALSO SEEN IN SOME CASES. ONE OF THE SEVEN CASES WAS COMPLETELY ASYMPTOMATIC AND DIAGNOSED ON THE BASIS OF EPIDEMIOLOGIC FOLLOW-UP OF AN INDEX CASE.

5. THE INCUBATION PERIOD IS DIFFICULT TO ACCURATELY MEASURE, HOWEVER, IN THESE CASES, THE ONSET OF SYMPTOMS VARIED FROM WEEKS TO MONTHS AFTER LEAVING SWA.

6. A SERUM INDIRECT IMMUNOFLUORESCENT ANTIBODY (IFA) TEST IS AVAILABLE AT WALTER REED ARMY INSTITUTE OF RESEARCH (WRAIR) THROUGH THE WALTER REED ARMY MEDICAL CENTER, HOWEVER, THERE IS NO COMMERCIALLY AVAILABLE SEROLOGIC TEST CURRENTLY AVAILABLE IN THE UNITED STATES TO CONFIRM INFECTION. WITH THIS TEST, IN PATIENTS WITH SUGGESTIVE SIGNS AND SYMPTOMS OF THIS LEISHMANIA INFECTION, A SERUM IFA EQUAL TO OR GREATER THAN 1:32 SUGGESTS INFECTION. DIAGNOSIS CAN BE CONFIRMED BY IDENTIFYING THE ORGANISM IN TISSUE AN CULTURING A BONE MARROW ASPIRATE. SMEARS OF THE ASPIRATE WERE NEGATIVE BY GIEMSA STAIN, BUT THE ORGANISM WAS DETECTED AT WRAIR USING ANTI-LEISHMANIA MONOCLONAL ANTIBODIES TAGGED WITH FLUORESCEIN.

7. THE TREATMENT OF CHOICE IS SODIUM STIBOGLUCONATE (PENTOSTAM), AN INVESTIGATIONAL NEW DRUG (IND) PRODUCED BY WELLCOME TRUST OF GREAT BRITAIN. IT IS AVAILABLE UNDER PROTOCOL TO MILITARY PHYSICIANS AT WALTER REED ARMY MEDICAL CENTER THROUGH WALTER REED ARMY INSTITUTE FOR RESEARCH, WASHINGTON, DC AND TO CIVILIAN PHYSICIANS FROM THE CENTERS FOR DISEASE CONTROL (CDC), ATLANTA, GEORGIA.

8. FIVE OF THESE SEVEN CASES WERE TREATED WITH SODIUM STIBOGLUCONATE. IN TWO OF THESE FIVE CASES, TREATMENT WAS DISCONTINUED EARLY BECAUSE OF THROMBOCYTOPENIA. FOLLOWING TREATMENT, ALL FIVE PATIENTS RECOVERED, RETURNED TO DUTY AND ARE BEING FOLLOWED. A SIXTH CASE WAS ASYMPTOMATIC, THEREFORE, NOT TREATED WITH SODIUM STIBOGLUCONATE AND IS BEING FOLLOWED CLOSELY. THE SEVENTH AND MOST RECENT CASE IS STILL UNDER CARE AT WALTER REED ARMY MEDICAL CENTER.

9. THE MILITARY MEDICAL DEPARTMENTS ARE ACTIVELY INCREASING THEIR CASE FINDING EFFORTS BY HEIGHTENING AWARENESS OF BOTH MILITARY AND CIVILIAN PHYSICIANS TO THIS CLINICAL SITUATION. SINCE MANY OF OUR MILITARY PERSONNEL WHO DEPLOYED TO THE GULF WERE RESERVISTS WHO, IF ILL, WOULD VISIT THEIR PERSONAL CIVILIAN PHYSICIAN IN THE PRIVATE SECTOR, THE CDC AND FDA ARE PARTICIPATING IN THIS EFFORT.

10. TRANSMISSION OF THE ORGANISM IS BY THE BITE OF A SANDFLY. MAN IS AN INCIDENTAL HOST. PERSON-TO-PERSON TRANSMISSION OF THIS FORM HAS NOT BEEN REPORTED.

11. THERE HAVE BEEN FIVE CASES REPORTED IN THE WORLD LITERATURE OF VISCERAL LEISHMANIASIS (L. DONOVANI) TRANSMITTED THROUGH BLOOD TRANSFUSIONS. NO CASE OF TRANFUSION-ASSOCIATED TRANSMISSION HAS BEEN REPORTED WITH L. TROPICA BUT A THEORETICAL RISK EXISTS. THEREFORE, AS A CONSERVATIVE SAFETY PRECAUTION DUE TO THIS THEORETICAL RISK AND THE ABSENCE OF A SIMPLE SCREENING TEST, THE ASSISTANT SECRETARY OF DEFENSE (HEALTH AFFAIRS) HAS DIRECTED MILITARY SERVICES TO DEFER UNTIL FURTHER NOTICE ALL PERSONNEL FROM BLOOD DONATION WHO WERE STATIONED IN SAUDI ARABIA, KUWAIT, IRAQ, BAHRAIN, QATAR, UNITED ARAB EMIRATES, OMAN AND YEMAN FROM 1 AUGUST 1990 AND THEREAFTER.

12. BLOOD PRODUCTS ALREADY COLLECTED FROM GULF WAR RETURNEES CURRENTLY IN THE INVENTORY ARE NOT BEING WITHDRAWN BECAUSE OF THE VERY LOW RISK OF CONTAMINATION BALANCED AGAINST THE RISK OF A SUDDEN SHORTFALL OF CRITICAL BLOOD SUPPLIES. UNDER CURRENT PROCEDURES, DONORS WHO WERE SYMPTOMATIC OR BLOOD UNITS WITH AN ELEVATED ALT LEVEL WOULD HAVE ALREADY BEEN EXCLUDED. NONE OF THE IDENTIFIED CASES HAS DONATED BLOOD AND ALL PATIENTS WITH ANY FORM OF LEISHMANIASIS ARE PERMANENTLY DEFERRED FROM FUTURE DONATIONS. A BLOOD DONATION HISTORY WILL BE TAKEN FROM ANY FUTURE CASE AND, IF POSITIVE, A COMPLETE LOOKBACK FOR THAT CASE WILL BE CONDUCTED.

13. THE POTENTIAL THREAT OF EXOTIC PATHOGENS IN SOUTHWEST ASIA WAS REVIEWED BY MILITARY MEDICAL PLANNERS PRIOR TO THE GULF WAR AND IS OUTLINED BY THE NEW ENGLAND JOURNAL OF MEDICINE ARTICLE PUBLISHED ON 21 MARCH 1991 BY GASSER, ET AL, AND SUPPLEMENT NUMBER 3 TO REVIEWS OF INFECTIOUS DISEASE, JANUARY 1991 BY OLDFIELD, ET AL. THESE ARE EXCELLENT REFERENCES FOR PRACTICING PHYSICIANS.

14. ANY PHYSICIAN WHO SUSPECTS LEISHMANIASIS INVOLVING DEPARTMENT OF DEFENSE SERVICE PERSONNEL SHOULD CONTACT THE APPROPRIATE SERVICE INFECTIOUS DISEASE CONSULTANT.

U.S. AIR FORCE: GREGORY MELCHER, MD
MAJ, USAF
WILFORD HALL MEDICAL CENTER
LACKLAND AIR FORCE BASE

BT
UNCLAS FINAL SECTION OF 02
SAN ANTONIO, TX 78236
COM. TEL: (512) 670-7444
DSN: 554-7444
FAX: (512) 675-0173

U.S. NAVY/MARINES: EDWARD OLDFIELD, MD
CAPT, USN
NAVAL HOSPITAL
SAN DIEGO, CA 92134
COM. TEL: (619) 532-7475
DSN: 522-7475
FAX: (619) 532-7484

U.S. ARMY: CHARLES N. OSTER, MD OR ALAN MAGILL, MD
COL, USA MAJ, USA
INFECTIOUS DISEASE SERVICE
WALTER REED ARMY MEDICAL CENTER
WASHINGTON, DC 20307
COM. TEL:(202) 576-0585/0588
DSN: 291-0585/0586
FAX: (202) 576-2478

WE RECOMMEND THAT, FOR OTHER INDIVIDUALS, THE CDC BE CONTACTED
CENTERS FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333
DRUG REQUEST: (404) 639-3670 (MONDAY-FRIDAY)
OR
(404) 639-2888 (EVENING, WEEKEND, HOLIDAY)

ACTION OASG(3) (W)
INFO RETURN TO WRO(1) DAAR(3) SCB REVIEW(1)

MCN=91317/25435 TOR=913172/1609Z TAD=91317/1633Z CDSN=MA0334
ARMY SECTIONAL MSG
PAGE 1 OF 2
R 1218532 NOV 91
02 SECT MSG

REQUEST IMMEDIATE DISSEMINATION TO ALL APPROPRIATE PERSONNEL TO INCLUDE ALL CLINICIANS AND BLOOD BANKERS.

POINTS OF CONTACT ARE COL PEAKE, DSN: 289-0141 OR (703) 756-0141, COL ERDTMANN, DSN: 289-0125 OR (703) 756-0125; COL TOMLINSON, DSN: 289-0135 OR (703) 756-0135, OFFICE OF THE SURGEON GENERAL. BT

DEPARTMENT OF THE ARMY
OFFICE OF THE SURGEON GENERAL
5109 LEESBURG PIKE
FALLS CHURCH, VA 22041-3258

REPLY TO ATTENTION OF

SGPS-PSA 26 May 1992

MEMORANDUM FOR Chairman, Leishmaniasis Committee

SUBJECT: Minutes of the Administrative Subcommittee of the Triservice Committee on Surveillance and Control of Leishmania among Gulf War Returnees

1. This committee meeting was held at 1430 hours, 22 May 1992, in Room 674, Skyline 6, Office of the Army Surgeon General. Attendees were:

NAME	Organization	Telephone/Fax
COL C. Oster	WRAMC	202-576-0587/
COL P. Tomlinson	USA OTSG PM	703-756-0124/0163
COL W.E. Savage	NGB	703-756-2799/4731
COL D. McKnight	USA OTSG NG Liaison	703-756-8048/
LTC R.B. Chapman	USA OTSG PAD (Chairman)	703-756-0102/0163
LTC S. Scanlon	USA OTSG USAR Liaison	703-756-8056/0243
MAJ P. Marcieski	USA OTSG PAD	703-756-0105/0163
MAJ R. Lipnick	USA OTSG PM	703-756-0357/0163
MAJ M. Coleman	USA NGB	703-756-7772/1225
CPT M. Poulsen	USAF Health Care Ops	202-767-5066/6208
MSG N. Rodriguez	USA OCAR	703-696-2965/226-5300
Ms. K. Posey	DVA	202-535-7375/
Ms. L. Hawkins	VA Med Center,Wash,DC	202-745-8248/
Ms. T. Wortzel	USA OTSG Med Standards	703-756-0150/0163
Ms. C. Vannerman	USA OTSG Med Standards	703-756-0157/0163
Mr. L.C. Procter	USA NGB	703-756-0752/4731
Ms. V. Stephanakis	USA OTSG Public Affairs	703-756-8022/4670

2. Business:

a. LTC Chapman opened the meeting, welcomed and thanked the attendees for their interest and support by attending this meeting, and distributed a packet of historical documents (Encl 1) containing clinical and administrative information regarding Leishmaniasis.

b. He stated the purposes of the meeting.

(1) To evaluate the effectiveness of the procedures put in place in December 1991 to identify, evaluate and treat SM suspected of having Leishmaniasis.

(2) To refine these procedures as necessary, and emphasize "getting the word out" to potentially infected SM.

SGPS-PSA
SUBJECT: Minutes of the Administrative Subcommittee of the Triservice Committee on Surveillance and Control of L.tropica among Gulf War Returnees

b. COL Oster presented an informative summary of the various types of Leishmaniasis and the connection with Operation Desert Shield/Storm.

He then presented several problems reported to him by SM who were eventually referred to WRAMC, and the difficulties they encountered getting into, and through the system.

COL Oster also proposed that a clinical algorithm be developed to standardize the initial evaluation of suspected SM, which will assist primary care clinicians, and the Infectious Disease Service staff at WRAMC, to initially identify and evaluate potential Leishmaniasis patients. This would also allow initial evaluation and follow up to be conducted at the local level where it would be more convenient for the patient. This would also assist the staff at WRAMC manage the cases more efficiently and effectively.

COL Oster suggested that the algorithm be patterned after the "Hepatitis C Program" procedures. After much supportive discussion, it was recommended that COL Oster pursue this recommendation for an algorithm. If approved by all necessary parties, to include OASD Health Affairs, then dissemination of this algorithm, with additional instructions, would be made throughout the country.

c. Ms. Posey then informed the committee that the VA was in the process of obtaining approval to implement a Registry of Desert Storm Veterans as these patients present themselves for care at a VA facility. This was of interest to the DOD attendees as a potential source of identifying SM with other than Leishmaniasis. It was noted, however, that the patients that were being followed at military facilities would not be in the VA registry, although eventually would become DVA beneficiaries.

COL Oster stated that WRAMC and WRAIR are in the process of developing a registry for patients evaluated at WRAMC. It would be desired to share this information with the DVA.

Ms. Posey gave the names and telephone number of the Chief, Infectious Disease at the VA Medical Center in Washington D.C., Dr. Fred Gordon, and his assistant, Dr. Sacks, to COL Oster for future contact(202-745-8301) and information sharing.

d. Ms. Hawkins asked if it were possible for the DVA patients that are referred to WRAMC to stay at the VA Medical Center in Washington, D.C. while they are being evaluated/treated at WRAMC. COL Oster replied that this may work to a limited degree with some patients , however the patients' responses to some tests and treatment could leave them to ill for transferring back and forth.

SGPS-PSA
SUBJECT: Minutes of the Administrative Subcommittee of the Triservice Committee on Surveillance and Control of L.tropica among Gulf War Returnees

e. LTC Chapman then asked everyone to re-energize their respective commands and agencies to be alert to the SM who are yet to present themselves for care. It is critical that these SM, once identified, are evaluated according to the instructions of the staff at WRAMC. If required, units, ARPERCEN, the NGB, the DVA, need to facilitate timely referral of these SM to WRAMC.

It was decided that the guidance and procedures presently in place would be emphasized and followed. Additional guidance would wait upon a decision regarding the acceptance and implementation of a clinical algorithm to address the initial evaluation of Leishmaniasis.

It was further decided that a Public Affairs approach to "getting the word" out would not be advisable as the symptoms of Leishmaniasis were vague and general and that SM would unnecessarily worry, wrongfully refer themselves for evaluation.

f. The recent decision of the Office of the Assistant Secretary of the Army, dated 21 May 1992, offering Army Reserve Component soldiers with symptoms of Leishmaniasis, to be placed back on active duty for the duration of the acute phase treatment was briefly discussed. Procedures to implement this decision were yet to be worked out.

3. Summary of actions to be taken:

a. Reemphasize to all appropriate commands and agencies the procedures to identify and assist SM who present with symptoms of Lieshmaniasis with the evaluation and treatment process, and to administratively assist the SM with necessary orders and other entitlements. ACTION: All Attendees.

b. Develop a clinical algorithm for review, necessary approvals, and implementation throughout the U.S. which will standardize the initial evaluation of SM who present themselves with symptoms of Leishmaniasis. ACTION: COL Oster.

4. The meeting adjourned at 1600 hours, 22 May 1992.

R. Bernard Chapman Jr.

Encl

R. Bernard Chapman, Jr.
LTC, MS
Asst. Chief, Patient
Administration Division

LEISHMANIASIS MEETING

22 MAY 1992

AGENDA

1. 1430 - a. Welcome. LTC Chapman

b. Purposes.

* To evaluate the effectiveness of the procedures put in place Dec 91 to identify, evaluate and treat SM suspected of having Leishmaniasis.

* To refine procedures as necessary, and empnasize "getting the word out."

c. Introductions

2. Background development of current policy for identifying and evaluating SM suspected of having Leishmaniasis. LTC Chapman

3. Summary of feedback from SM who have been identified and "processed through the system" to be evaluated at WRAMC. COL Oster

4. Discussion. All

5. Adjournment. LTC Chapman

MEMORANDUM FOR L TROPICA WORKING GROUP 5 MAY 92

MEMORANDUM FROM COL BERMAN

SUBJECT: Minutes of 9th meeting--4 May 92

1. Attendees were: COL Oster, COL Sadoff, COL Berman (Chair),LTC Perkins, LTC Ballou, MAJ Nuzum, MAJ Magill, MAJ Grogl, Dr. Nacy, Dr. Leiby.

2. Chair's report:

COLs Berman, Schuster, Salvado presented the program to MRDC on 17 Apr 92. The diagnostic initiatives IFA, ELISA, Western Blot, PCR were approved by MRDC to approximately the amounts on the appended spreadsheet. Skin test was not approved, because of the concern that it might require too long (1 1/2 yrs) to produce a product. Work on leishmania survival in blood was approved, to the amount shown on the spreadsheet, but tropism of New and Old World Leishmania was not approved. Patient diagnosis on new cases, data management, sample shipping, and consultation was approved to the amount shown.

The working group thought that a skin test was as likely as any other diagnostic test to be useful in the ODS patients and that funding for the test should be approved. The working group also thought that tropism of Leishmania tropica to blood (as opposed to tropism of Leishmania in general to organs in general) was as relevant to the management of the ODS patients as the approved work on Leishmania survival in blood, and should be approved for funding.

3. Diagnostic Initiatives:

a1) IFA-slide test: The IFA initiative was divided up into the IFA slide test and a new diagnostic modality, the FACS test.

The IFA slide test was used on paired samples, originally collected for a survey of tick-borne diseases, from 209 ODS troops. 11 paired samples showed a 4-fold rise from pre to post deployment. No sample showed an 8-fold or higher rise; there were approximately 53 samples with a 2-fold rise. [None of 49 (?) Marines on board a ship showed a 4-fold rise]. If we assume that a 4-fold rise is diagnostic of infection, then 5% of this ODS unit was infected with Leishmania.

a2) IFA-FACS: Deliberately infected monocytes are fixed with paraformaldehyde, permiabilized with saponin, reacted with fluorescent anti-leishmania antibody, and subjected to flourescent activated flow cytometry. Infected monocytes are more fluorescent than uninfected monocytes, and one infected monocyte can be detected in the midst of 33,000 uninfected monocytes. This technique shows promise for the detection of infected monocytes in blood.

b,c) ELISA: LTC Martin (USAMRU-K) grows Leishmania in protein free media, then collects the media which contains Leishmania-excreted proteins. These proteins can be used on a Western blot, and bands recognized by patient's sera can be eluted. The eluted bands can be used in an ELISA. This procedure works well for L donovani: excreted, eluted bands are the basis for a successful ELISA that recognizes serum from L donovani infected patients, but does not recognize sera from L tropica cutaneously infected patients. LTC Martin is now attempting the same approach to originating an ELISA using L tropic excreted factor that recognizes the serum of our viscerotropic L tropica patients. LTC Martin will be TDY to WRAIR in ~June 92 to demonstrate his results and procedures.

d) PCR: responses to RFP are being considered,

e) Skin test:

1. CDI proposes contracting for 1000 doses of L tropica skin test material for $71K. If this material has only slight reactivity (defined as <3 mm induration) in normal volunteers (80K test), then CDI proposes to GMP manufacture 500,000 doses in-house in a new GMP facility for $300K. The contracts for all these procedures require about 12 weeks. If funding is received by 1 Jun 92, most contracts can probably be let by Sep 92 (although work will not start immediately in all contracts). Some contracts can not be let by the deadline of Sep 92: this work will have to be performed in house gratis.

2. Wellcome has been contracted about their L major antigen, presently being used in the US

ENCL 3

by Dr. Neva. There is no more material at Wellcome; Wellcome has no intention of making more; the facility in which the material was made is no longer GMP and will not be upgraded.

3. The WHO stated that their L major antigen is being produced at the Pasteur Institute, Teheran, and that there are large amounts (100,000 doses) available for a nominal price (1K for 10,000 doses). This antigen is presently being used by the NIH with NIH human use approval but without an IND. For MRDC/HSC to use this material, it would probably have to go through sterility/lack of acute toxicity tests (2K).

4. Plan: The L major antigen should be compared to the L tropica antigen made in 1000 doses in preclinical tests (5K extra) and in normal volunteers. Comparison of L major antigen to L tropica antigen therefore costs only 7K extra. This is money well spent to see if there is an alternative to spending 300 K to make 500,000 L tropica doses. However, if the L. tropica antigen is superior, the 300K mentioned above to make 500,000 doses would be money well spent.

The major problem with this Plan is that I doubt that with the several in vivo tests needed (preclinical animal test, human phase I tests at a DoD facility, administration to patients throught the Army, Navy, and Air Force), someone somewhere will not require an IND. COL Schuster and I have not yet devised an ironclad means of getting assurances ahead of time that an IND will not be required.

4. Other initiatives:

f2) Survival in blood: The recommendations to freeze blood are the same as the recommendations to freeze Leishmania, such that they will remain viable.

g) Patient diagnosis: It was briefly reported that two new cases (one visceral, one cutaneous) have been diagnosed.

k) Extent of infection in the force: 26 ODS members of Foreign Material Intelligence Branch, now at Aberdeen, were subjected to L major skin test by Dr. Neva. 3 had >5 mm induration; the accepted criteria for Leishmania skin test positivity. 5 others had 3-4 mm induration. [We await data on the extent of induration in US controls that could not have been exposed to Leishmania.] This gives a ~10% or 35% incidence of infection in this small force.

5. Funding:

a) MRDC: Of the $3400K that arrived from OTSG to MRDC, $2540K was passed to WRAIR. The assumption is that MRDC needed 860K to pay for the PCR RFP (?550K) and the 154K for the terminated IFA testing at Salk. It is hoped that MRDC will also pay the 77K for Reed's contract to try to devise a specific ELISA antigen. If so , that would leave~80K at MRDC. Berman will call Hill to discuss.

b)Approved for WRAIR: Of the 2540K that arrived at WRAIR, the accompanying spread sheet [May 92] shows that 1250 is needed for past expenditures made upon the promise of funds.

In addition, 426 is needed for future expenditures approved by MRDC, and 282 is needed for future salary costs approved by MRDC. Future costs represent the total amount needed until Sep 93, which is apparently the last date that ODS funds, obligated in FY 92, can be utilized. The salaries are MIPRed to the AFIP, which has an agreement with the ARP to provide non-personal services assistance. MAJ Horning will call AFIP to see how much of previous MIPR is as yet unspent, and to verify that 12 more months of services can be contracted in Sep 92.

If WRAIR adds a 16% overhead charge to all new WRAIR expenditures (426 + 282), the result is 1250 + 524 + 336 = 2110. WRAIR received 2540.

It is suggested to Dep Dir/ Dir Resource Management that the 1250 be paid off, that the Future Exp amounts indicated without italics on the spreadsheet (440 = 524 with 16% overhead) be put into the funding accounts of the respective divisions, and that 336 (282 with 16% overhead) be MIPRed to the AFIP. WRAIR would still have ~300 unspent. The unspent amount would be larger if MAJ Horning can recover that percent of the original 250K given to the AFIP that hasn't yet been spent.

c) Requested by WRAIR: Since the grand total of approved expenditures, requested expenditures, and 16% WRAIR overhead equals 3784K, if the requests to fund development of a skin test (452 K) and to further examine Leishmnania blood interaction (174 K) are granted, the total MRDC-WRAIR requirements would be ~400K more than the 3400K from OTSG, and ~600K more than the present amount spendable by MRDC/WRAIR.

COL Sadoff will circulate the proposal to develop a skin test, and send the proposal to RAD 1. COL

Berman will do the same for the Leishmania-Blood studies. If RAD1 and MRDC comptroller approve of these two items, MRDC will have to request ~400K from OTSG.

		WRAIR	COSTS	($K)	[May 92]	
	PREVIOUS	*FUTURE EXP*	*FUT SALAR*	*CONTRACT*	*CONTRACT*	*OTHER*
INITIATIVE	*(TOTAL)*	*(TOTAL)*	*(TOTAL)*	*(TOTAL)*	*NOTES*	*NOTES*
1. DIAGNOSTIC TESTS						
a1. IFA slide test		32 (ET)	0	154	Salk	OK: APR 92
a2. IFA-FACS		36 (CDI)	0	0		
b. ELISA		72(CDI)	0	77	Reed	OK: APR 92
c. Western		0	90(1: CDI)	0		OK: APR 92
d. PCR		10 (ET)	24(0.5:ET)	550	RFP	OK: APR 92
e. Skin test-1000 dose		*0*	*0*	*72*	*via CDI*	*requested*
e. Skin test-500000		*300 (CDI)*	*0*	*80*	*via CDI*	*requested*
2. LEISH--BLOOD						
f1: TROPISM TO BLOOD		*36(CDI)*	*0*	*120*	*AFIP*	*requested*
f2: SURVIVAL IN BLOOD		36(ET)/36(CDI	0	0		OK: APR 92
f3: TRANSFUSE FROM BLOOD		*18(ET)*	*0*	*0*		*requested*
3. PATIENT DIAGNOSIS						
g: NEW CASES		170 (ET)	168(2.5:ET)	0		OK: APR 92
l:DATA MANAGEMENT		0	0	0		OK: APR 92
m: SAMPLE SHIPPING		24 (ET)	0	0		OK: APR 92
n: CONSULTATION		24 (HQ)	0	0		OK: APR 92
						GRAND
TOTALS (no req)	1250	440	282	781		2753
TOT (+16% WRAIR)	1250	524	336	781		2891
TOTALS (includes requests+16% WR)	1250	945	336	1053		3584

ASBPO
24 April 1992

POSITION PAPER

SUBJECT: Rescission of Leishmania Donor Deferral Policy

BACKGROUND:

1. On 12 November 1991, the ASBPO, at ASD(HA)'s request, issued a policy letter requesting deferral of all donors who served in the Persian Gulf until sufficient evidence could be amassed to modify that policy or until a screening test could be developed that would protect the blood supply.

2. Subsequently, the major civilian blood collection agencies also published a similar policy, but indicated that the deferral would be in effect until 1 January 1993. A firm date was assigned by the civilian community to encourage donors to return and to not discontinue donating indefinitely.

3. Both the military and civilian blood collecting communities intended to publish additional information well in advance of the 1 January 1993 date indicating necessary deferral policy changes.

4. A U.S. Navy Surgeon General memorandum, dated 16 April 1992, was sent to the DASD(HA)PA/QA requesting that the leishmania donor deferral policy be rescinded. It cited that the first 22 cases of leishmania had already been identified when the initial deferral policy was published and that there were only three additional cases identified as of 4 March 1992. The infection rate was stated to be only 0.005 percent.

RECOMMENDATION:

Since it has only been two months since the last case of leishmania was diagnosed, recommend that an additional two months of no new cases pass before rescinding the policy. In addition, the Leishmania Working Group should develop guidelines for deferral of those individuals who have served in the Persian Gulf area since completion of Operation DESERT STORM. These guidelines should be coordinated with civilian transfusion-transmitted disease experts to ensure uniformity of policy among all U.S. blood collecting agencies.

Lt Col M.J. Ward
(703) 756-8024

CLINICAL COURSE (VM)

MARCH 1991	RETURN FROM ODS
28 AUG 1991	SEROLOGY SCREEN OF UNIT AT FT. BRAGG LEISHMANIA IFA TITER OF 1:32, PATIENT ASYMPTOMATIC
LATE OCTOBER 1991	ONSET OF ILLNESS OVER SEVERAL DAYS MALAISE, FATIGUE, ARTHRALGIAS, MYALGIAS ANOREXIA, NAUSEA, NONSPECIFIC ABDOMINAL PAIN HEADACHES, DIZZY, NONPRODUCTIVE COUGH TENDER RUQ AND LUQ
13-18 NOV 1991	FIRST WRAMC ADMIT
14 NOVEMBER 1991	FIRST BONE MARROW ASPIRATION 3 AMASTIGOTES SEEN ON MONOCLONAL GIEMSA AND CULTURE NEGATIVE PCR OF BLOOD AND MARROW (+) FOR LEISH
6-16 JAN 1992	SECOND WRAMC ADMIT
07 JANUARY 1992	SECOND BONE MARROW ASPIRATION MONOCLONAL/GIEMSA/CULTURE NEGATIVE PCR OF BLOOD (+) FOR LEISH LEISH SKIN TEST (-), T CELL PROLIFERATION (+)
19 FEB TO PRESENT	THIRD WRAMC ADMIT HEPATOMEGALY FIRST NOTED ON PE
25 FEBRUARY 1992	EGD + BX = MILD GASTRITIS WITH ULCER AT GE JUNCTION
27 FEBRUARY 1992	CT OF CHEST NORMAL (? OF PERIHILAR ADENOPATHY ON CXR). OLD GRANULOMATOUS DZ
02 MARCH 1992	CT OF ABDOMEN - ? OF MASS IN TAIL OF PANCREAS, SPLENIC GRANULOMAS, AND HEPATOMEGALY

10 MARCH 1992	LIVER BX = MILD CHRONIC PORTAL TRIADITIS AFB AND FUNGAL SMEAR/CULTURE NEGATIVE MONOCLONAL SMEAR POSITIVE SUSPECT STRUCTURES SEEN ON H&E BUT NOT DX FOR AMASTIGOTES CULTURE NEGATIVE FOR LEISHMANIA
01 APRIL 1992	REPEAT CT OF ABDOMEN - UNCHANGED. PROBABLY NORMAL ANATOMIC VARIANT BUT CANNOT R/O MASS IN TAIL.
07 APRIL 1992	DEDICATED CT OF PANCREAS WITH REALTIME CONTRAST ENHANCEMENT SHOWS NORMAL PANCREAS.
08 APRIL 1992	LEFT AXILLARY LYMPH NODE EXCISIONAL BX CULTURE POSITIVE FOR PROMASTIGOTES AT 8 DAYS. ISOLATE CHARACTERIZED AS L.TROPICA.

SUMMARY OF DX TEST RESULTS

STOOL O&P AND C&S NEGATIVE FOR ENTERIC PATHOGENS/PARASITES
STRING TEST FOR GIARDIA NEGATIVE
MONOSPOT NEG, VCA-IGM NEG, VCA-IGG=1:640, EA=1:40, EBNA(+)
CMV IGM NEGATIVE, CMV IGG (+)
HTLV-1, HTLV-2 NEGATIVE. HIV ANTIBODY NEGATIVE X 2 (NOV91,MAR92)
NEGATIVE RPR, B. CANIS, B. ABORTUS, TOXO, F. TULAR. SEROLOGY
NORMAL ESR
NORMAL TFT'S AND CORTROSYN STIM TEST (7 TO 34)
AFB/FUNGAL SMEAR AND CULTURE OF LIVER, 2ND BONE MARROW, LYMPH NODE NEGATIVE, ALL SPUTUM SPECIMENS AFB SMEAR NEGATIVE BUT 1 OF 3 CULTURE (+) FOR M. GORDONAE.
PPD NEGATIVE WITH NORMAL DTH RESPONSE

LEISH SPECIFIC

T CELL PROLIFERATION FOR LEISH AG (+)
PCR of peripheral blood (+) X 2
LEISH SKIN TEST NEGATIVE X2 (JAN AND MAR 92)

SUMMARY

35 year old airborne trooper with subacute onset of clinical symptoms 7 months after return from ODS. Clinical presentation was a nonspecific systemic illness that evolved into a chronic fatigue syndrome. RUQ tenderness with hepatomegaly, LUQ tenderness but no palpable spleen, and intermittent peripheral adenopathy were seen during the course of illness. No anemia, leucopenia, thrombocytopenia, elevated ESR, or elevated transaminases were ever documented. Modest hypoproteinemia and hypoalbuminemia were seen. Modest lymphopenia and monocytosis were also seen.

The patient had 2 bone marrow aspirations, 1 liver biopsy and a lymph node biopsy before a Dx was unequivocally established. In addition, multiple radiographic studies and blood tests were performed. Patient endured 5 months of illness prior to receiving potentially curative RX.

As of 17 April, patient has had 75 inpatient days at WRAMC. He will require a month of Rx and a month of convalescent leave prior to return to duty if no complications occur. Total loss of time to his unit to date is almost 6 months with a minimum of 2 more months to go.

Alan J. Magill M.D.

UPDATE:

Patient has completed 22 of a planned 30 doses of Pentostam. He has had all the usual side effects of this drug to include drug associated pancreatitis. However, his laboratory abnormalities and abdominal symptoms have improved with continued therapy and we anticipate completing the full course of treatment.

06 May 1992
Alan J. Magill M.D.

WRAMC Leishmania Update for 06May92 OTSG Meeting

1. One new case of viscerotropic leishmaniasis due to L.tropica.

   Case totals 10 Viscerotropic
   17 Cutaneous
   27 total ODS related Leishmaniasis

2. Discussion of lessons learned from latest case

3. Current WRAMC inpatient census

   4 patients currently being treated with Penstostam
   2 cases of cutaneous - one ODS related and one from French Guiana.
   2 cases of ODS related viscerotropic disease

   5 others for Dx evaluation
   4 suspect viscerotropic cases
   1 suspect cutaneous case

4. Results of APG skin test survey

Alan J. Magill M.D.

AGENDA

Triservice Committee on Surveillance and Control of L.tropica Among Gulf War Returnees

29 May 1992
Room 535--Skyline 5
Falls Church, Virginia 22041-3258

I. Approval of the Minutes

II. Old Business

Issues	Action
Leishmaniasis Resourcing Update	MAJ McMaughan
Patient Inquiries/Clinical Status/New Cases Update	MAJ Magill
Status of DOD Blood Donation Deferral	LTC Ward
Patient Access	LTC Chapman
Review Results of WRAIR Special Working Group	COL Berman

III. New Business

Open Discussion	Committee Members

DEPARTMENT OF THE ARMY
U S ARMY HEALTH FACILITY PLANNING AGENCY
5109 LEESBURG PIKE
FALLS CHURCH, VA 22041-3258

REPLY TO
ATTENTION OF

SGPS-CP

MEMORANDUM FOR See Distribution

SUBJECT: Minutes of the Triservice Committee on Surveillance and control of L.tropica among Gulf War Returnees

1. The ninth ad hoc committee was held at 1400 on 5 May 1992, in Room 674, Skyline 6. Attendees were:

Name	Organization	Telephone	FAX Extension
COL Berman	WRAIR-MRDC	202-576-3453	x3114
COL Claypool	SGPS-CP-M	703-756-0148	x0163
COL Peterson	OASD (HA)		
COL Shuster			
COL Wright			
CDR Cunnion			
CDR Parsons			
COL Oster			
COL Tomlinson	SGPS-PSP	703-756-0128	x0163
LTC Brown	SGPS-CP		
LTC Gasser	USAF		
MAJ Grogl	WRAMC		
MAJ Magill	WRAMC	202-756-1740	
MAJ Lewis	SGPS-CP	703-756-0160	x0163
MAJ Lipnick	SGPS-PSP	703-756-0123	

2. The minutes of the previous meeting were approved.

3. Old Business:

a. Colonel Oster asked about WRAMC resources. The committee advised him to have the WRAMC Comptroller contact Major McMaughan, DASG-RMZ.

b. A clinical leishmania update was provided and discussed by Major Magill (Encl 1).

c. The challenges of patients suspected of leishmaniasis getting into the established protocols was briefly discussed. Major Marcheski will give an update at the next meeting.

d. A position paper on the donor deferral policy was presented (Encl 2).

e. Colonel Berman provided an update on the current status of the L. Tropica Working group (Encl 3).

4. Summary of Actions:

Issues	Action
Leishmaniasis Resourcing Update	MAJ McMaughan
Patient Inquiries/Clinical Status/New Cases Update	MAJ Magill
Patient Access	LTC Chapman
Status of DOD Blood Donation Deferral	LTC Ward
Review Results of WRAIR Special Working Group	COL Berman

5. There being no further business, the meeting was adjourned with the next meeting scheduled for ~~1400~~ 1000, on 29 May 1992, in Room 535, Skyline 5 (Encl 4).

4 Encls
as

ROBERT G. CLAYPOOL
COLONEL, MC
Chief, Clinical Policy
Consultants Division

DISTRIBUTION:

COL Bancroft	USAMRDC	LTC Chapman, Jr.	SGPS-PSA
COL Berman	WRAIR-MRDC	LTC Roberts	SGPS-PSP
COL Claypool	SGPS-CP	LTC Ward	ASBPO
COL Hiner	SGPS-CP-P	LTC Wright	USAF/SGPA
COL Oster	WRAMC	MAJ DeFraites	WRAIR
CAPT Parsons	AFEB	MAJ Lewis	SGPS-CP
COL Robinson	WRAIR	MAJ Magill	WRAMC
COL Sprague	WRAMC Path	LCDR May	BUMED
COL Wear	AFIP	MAJ McMaughan	DASG-RMZ

MEMORANDUM FOR L TROPICA WORKING GROUP 28 May 92

MEMORANDUM FROM COL BERMAN

SUBJECT: Minutes of 10th meeting: 28 May 92

1. Chair's report:

Funding status: We are now entering the 3rd phase of the program. The first phase (Nov 91-Feb 92) was initial experiments and understanding the problems. The second phase (Feb 92-June 92) was obtaining authorization/funding for addressing the problems. The present 3rd phase is to actually address the problems.

Funds are authorized as per the appended memorandum for the record of 27 May 92. We have to spend the funds by Sep 92, and then produce data. Monthly progress reports are mandatory.

2. Diagnostic Initiatives: administration [ability to obligate funds] and science [data]

a,k) IFA: Future paired serum studies and extent of infection in force:

Having determined that on the basis of paired sera, a 4-fold rise in titers occurred in 5% of one unit and 0% of another unit, determinations will now be made of the incidence of 4-fold rises in other units. The only appropriate units are ones in which sera was drawn soon after the termination of ODS, so that ELISA titers do not have time to decline. There are 3 such units. The extent of infection in the force, on the basis of 4-fold rise in IFA titers, will then have been determined in 5 military units.

b) ELISA: no new data.

c)Western Blot.

LTC Martin (USAMRU-Kenya) has been growing Leishmania without added protein in the medium. This permits concentration of the parasite proteins that are excreted into the medium without the concentrate being overwhelmed with medium protein. When the medium from a 5-day culture of a USSR L tropica were concentrated by a factor of 10, run on a Western blot, and then reacted with a 1/500 dilution of post-deployment sera from 5 patients (4 confirmed cases and 1 highly suspected case), 2 patients (Ford/Loggins) showed a band at 55 kd. 3 patients (Molina/Boone/Patrick) do not show a band. All pre-deployment sera were negative, at 1/500 dilution. When the sera was used at a dilution of 1/50, the 55 kd band was recognized by sera from 3 other patients (Glass, Molina, Cullen) , but pre-depolyment sera were not run.

To determine if the excreted factors in 5-day old cultures of promastigotes maintained without protein have a 55 kd band recognized by patients' post-deployment but not pre-deployment sera [sensitivitiy], and not recognized by normal Americans [specificity]: MAJ Grogl will make available 4 L. tropica parasites representing all the excreted factor serotypes of the ODS patients; LTC Martin will grow the parasites and obtain the excreted factor; MAJ Klotz will run the Western blots against patient pre- and post-sera, against the pre-and post-sera of suspected cases, and on both theoretically non-exposed ODS members and on non-ODS Americans.

d) PCR: The PCR contract responses are still being reviewed by the Committee.

e) Skin test:

LTC Ballou presented a plan to develop an L tropica skin test antigen.

Stage1: MAJ Grogl and MAJ Magill will decide if WR 1063 or perhaps another strain should be the test organism, on the basis of clinical and lymphocyte transformation data. The several antigens will be tested by Dr. Leiby for potency in the L major infected C57Bl mouse model. The most potent antigen will then be forwarded to LTC Ballou for development. While the decision on an organism is being made, CBL labs in Baltimore, or perhaps Building 508, will receive a contract to grow parasites sufficient for 1000 skin tests, and to perform sterility/pyrogenicity/acute toxicity studies. Simultaneously, LTC Ballou will contact Dr. Brandt (USAMMDA) so that a task order for EER associates to prepare the IND can be awarded. The antigen, made under GMP by CBL or building 508, will be tested for potency under GLP by Dr. Leiby (Dept Cellular Immunology), and then submitted for phase I/II human testing at the WRAIR Clinical Center [contract already awarded]. In phase I/II testing, a range of doses [50 ug downwards] will be tested on both ODS cases and on normal volunteers.

Stage 2: If the 1000 doses made in stage 1 indicate that the material is appropriate in terms of lack of toxicity, sensitivity, and specificity, manufacture of up to 500,000 doses will ensue. On the assumption that stage 1 will proceed satisfactorily, Building 501 will be reconfigured for GMP manufacturing[150K]; media [20K] and vials [40K] will be purchased; culture flasks to prepare 45 liters [45,000 doses if there are 25 ug/10M organisms. 10M organisms per ml, and each skin test requires 25 ug] at a time will be acquired. If a source of 90K is located, fermenting equipment will also be purchased. Fermentation, if successful, will subject cultures to less contamination, and the space and time required for growing the cultures can at least be halved.

f) Leishmania blood studies:

Dr. Leiby continues to develop the IFA-FACS technique to recognize infection of blood monocytes with Leishmania. At present, one infected monocyte can be recognized in the midst of 30,000 uninfected monocytes. Future experiments will determine if blood can be manipulated [via Leukocyte-preparation tubes] to yeild a preparation that can be put into the FACS machine, the calculated limit of detection of infected monocytes spiked into blood; and whether patients with visceral leishmaniasis due to L donovani in Kenya, for whom at least 75% have organisms in the blood detectible by standard techniques, have demonstrable organisms in the blood detectible by FACS [positive controls for this technique].

Blood from patients with cutaneous disease and with viscerotropic L tropica disease may also be analyzed to determine if our patients have contamination of the blood with Leishmania. One problem may be that if an ODS patient's blood is positive for Leishmania by FACS, there will be no way to differentiate a false positive from a true positive because there is as yet no other technique as sensitive as FACS with which to compare the FACS data. One solution to this problem of false positivity is to buy (130K) a cell sorter for the FACS: the sorter removes the infected cells from the chamber after the infected cells are counted, permitting the putatively infective cells to be stained for visual identification of Leishmania.

g) New cases : One new case.

MAJ Magill is leaving the clinical service on 1 Jun 92, and there is as yet no designated replacement. For medical-legal reasons, the WRAIR Leishmania Section has a requirement to report to a clinician, and a need for an SOP concerning what paperwork should be filled out, prior to providing more clinical support to the WRAMC patients.

3. New business: Next meeting: Monday 6 Jul 92, 1200 hrs, building 40 [room TBA].

SGRD-UWM 27 May 92

MEMORANDUM FOR THE RECORD

SUBJECT: Funding for ODS Leishmaniasis Initiatives [CORRECTED]

1. The following are the agreed initiatives, performing divisions, and alloted expenses for the WRAIR ODS leishmaniasis program. These are the only funds which may be spent on this OTSG-funded program under the current guidelines of RAD 1 and the OTSG subcommittee. Modifications to this list must be approved by the undersigned.

2. APPROVED INITIATIVE	PERFORMING DIVISION	$K FUNDED
Diagnosis		
a) IFA	ET	32
	CDI	36
b) ELISA	Kenya	67
c) Western	CDI	72
d) PCR	ET	560
e) Skin Test	CDI	452 [72 not yet funded]
Blood Studies		
f1) Leishmania tropism	AFIP	120 [not yet funded]
f2) Leishmania survival	CDI	36
	ET	36
Patient Diagnosis		
g) New cases	ET	170
l) Sample Shipping	ET	24
n) Consultation	HQ	24
Previous P6 expenditures	HQ	1037
WRAIR overhead	HQ	295

3. The total funding of approved initiatives = $2769.
The total cost of unfunded approved initiatives = $192.
Since WRAIR received $3040, and the sum of the funded and unfunded initiatives = $2961, the unfunded approved initiatives can and will be funded.

4. Please (non-sequentially) affix signatures below and return to COL Berman for filing

WILLIAM H. BANCROFT
COL, MC
Director
Military Disease Hazards Research Program

AUGUST J. SALVADO
COL, MC
Director
WRAIR

BRAIN G. SCHUSTER
COL, MC
Division of Experimental Therapeutics
WRAIR

JONATHAN D. BERMAN
COL, MC
Chair, Leishmania Working Group
WRAIR

DS2 Health Hazards

- DS2 is a Mixture of Three Compounds.

  Health Effect Estimations Are Based on the Known Biological Activity of Each Compound.

- Four General Classes of Health Effects
  - Pulmonary and CNS Effects
  - Burns From Direct DS2 Contact
  - Allergic Manifestations
  - Reproductive Effects

DS2 HEALTH EFFECTS

- Health Hazard Data was Transmitted to DA Safety in February 1991.

  DA Safety Issued a Worldwide Safety of Use Message Within a Few Days.

- Propylene Glycol Monomethyl Ether (PGME) has Been Cleared by Toxicologists at the USAEHA as a Substitute in DS2 for Ethylene Glycol Monomethyl Ether (EGME).

  EGME is the DS2 Component Compound Linked to Potential Reproductive Toxicity.

DS2 HEALTH EFFECTS

Other Hazardous Components of DS2:

- Sodium Hydroxide
  - Caustic Alkali
    - Burns
    - Blindness
- Diethylenetriamine
  - Alkali Amine
    - Irritant Effects
    - Skin and Pulmonary Sensitization

DS2 HEALTH EFFECTS
FUTURE ISSUES

- Continued Hazard Communication
- Toxicity Clearances for Substitute Compounds
- Health Hazard Assessments for Future Decontaminating Systems

UNCLASSIFIED

P 5? [illegible]

[illegible]
CDRUSAREUR HEIDELBERG GE //[illegible]//
CDR USASDC HUNTSVILLE AL //CSSD-SD//
CINC FORSCOM FT MCPHERSON GA //[illegible]//
CDR TRADOC FT MONROE VA //ATOS//
CDR AMC ALEX VA //AMCSF//
CDRINSCOM FT BELVOIR VA //IAPER-MS//
CDRUSARPAC FT SHAFTER HI //APSL//
CDRUSACIDC WASHDC //CIPA-MPS-S//
CDR USAISC FT HUACHUCA AZ //ASPE-S//
CDR USARSC FT CLAYTON PN //SOSA-[illegible]//
CDRUSASOC FT BRAGG NC //AOPE//
SUPT USMA WEST POINT NY //MAPE-S//
COMUSARCENT RIYADH SA //[illegible] SAFETY//
INFO DA WASHDC //DAMO-SW/[illegible]//
CDRUSASC FT RUCKER AL //[illegible]//

UNCLAS
SUBJECT: SAFETY ALERT DECONTAMINATION SOLUTION 2 (DS2)
A. FM 3-5, 24 JUN 85, NBC DECONTAMINATION.
B. TB CML 113, 15 NOV 90, STORAGE AND SHIPMENT OF DS2.
1. DS2 IS THE MATERIAL OF CHOICE AS A DECONTAMINANT FOR CHEM AGENTS. THIS MESSAGE IS A REMINDER THAT SAFEGUARDS ARE REQUIRED WHEN USING AND/OR STORING DS2 TO ENSURE PERSONNEL SAFETY.
A. DS2 SHOULD NOT BE USED FOR TRAINING.
B. PERSONNEL HANDLING DS2 MUST USE PERSONAL PROTECTIVE EQUIPMENT. AS A MINIMUM, PROTECTIVE MASK AND RUBBER GLOVES MUST BE WORN. THIS DOES NOT CHANGE THE RESPONSIBILITY TO FOLLOW THE COMMANDERS GUIDANCE ON MOPP LEVEL. DECONTAMINATION OPERATIONS WILL BE CONDUCTED IN MOPP 4.
C. ENSURE DS2 CONTAINERS ARE STORED PROPERLY. DS2 AND SUPER TROPICAL BLEACH (STB) MUST BE SEPARATED FOR STORAGE AND TRANSPORT.
D. DO NOT USE DS2 AND STB TOGETHER. A FIRE COULD RESULT IF DS2 AND STB COME IN CONTACT.
2. POTENTIAL HEALTH EFFECTS OF EXPOSURE TO DS2 ARE:
A. DIRECTLY BREATHING DS2 MAY RESULT IN NAUSEA, VOMITING, AND RESPIRATORY TRACT IRRITATION. LENGTHY INHALATION EXPOSURE TO DS2 VAPOR MAY AFFECT REPRODUCTIVE ORGANS IN BOTH MALES AND FEMALES AS WELL AS HAVE EFFECTS ON PREGNANT FEMALES.
B. DIRECT SKIN CONTACT MAY RESULT IN BURNS.
C. DIRECT EYE CONTACT MAY RESULT IN [illegible]
D. DS2 MAY CAUSE ALLERGIC REACTIONS.
3. IN CASE OF EXPOSURE, SEEK MEDICAL ATTENTION IMMEDIATELY. EMERGENCY AND FIRST AID PROCEDURES ARE:
A. INHALATION: REMOVE TO FRESH AIR. IF BREATHING HAS STOPPED, GIVE ARTIFICIAL RESPIRATION.
B. SKIN CONTACT: FLUSH AWAY THE DS2 FROM THE SKIN WITH COPIOUS AMOUNTS OF WATER UNTIL "SOAPINESS" IS NO LONGER PRESENT.
C. EYE CONTACT: IMMEDIATELY FLUSH THE EYES WITH COPIOUS AMOUNTS OF WATER FOR AT LEAST 15 MINUTES.
D. INGESTION: IF THE PATIENT IS CONSCIOUS, GIVE AS MUCH MILK OR WATER AS TOLERATED. DO NOT REPEAT, DO NOT INDUCE VOMITING.
4. REQUEST DISSEMINATION OF THIS MESSAGE TO OPERATIONS, LOGISTICS, AND MEDICAL PERSONNEL AS APPROPRIATE.
5. HQDA POC IS MS. CONNIE [illegible], DAMS [illegible], DSN 225 [illegible]

ACTION OFFICER
[illegible] CONNIE [illegible]

[illegible]

UNCLASSIFIED

DEPARTMENT OF THE ARMY
U.S. ARMY CHEMICAL RESEARCH DEVELOPMENT AND ENGINEERING CENTER
ABERDEEN PROVING GROUND MARYLAND 21010-5423

REPLY TO ATTENTION OF

SMCCR-HV (40-5f)

13 September 1990

MEMORANDUM THRU Commander, U.S. Army Materiel Command, ATTN: AMCSG, 5001 Eisenhower Avenue, Alexandria, VA 22333-0001

FOR HQDA (SGPS-PSP), 5111 Leesburg Pike, Falls Church, VA 22041-3258

SUBJECT: Request for Toxicity Clearance for Reformulated Decontaminating Solution 2 (DS2)

1. Reference memorandum, SMCCR-PPP, 4 Sep 90, subject: Toxicities Assessment for New Formulation of Decontaminating Solution, DS2 Item

2. The current formulation for DS2 is 28% ethylene glycol monomethyl ether (EGME), 70% diethylene triamine, 2% sodium hydroxide. Efforts are currently underway to replace EGME with a less toxic substance, propylene glycol monomethyl ether (PGME), on a one for one basis.

3. Request a toxicity clearance be conducted to determine the potential toxicological effects of replacing EGME with PGME in DS2. Toxicological information on both compounds is provided at reference 1.

3. If further technical information is required it should be requested from Mr. Richard Forcheimer, SMCCR-PPP, DSN 584-5796

4. Point of contact for this office is Dr. Wade Kuhlman, [illegible] DSN 584-2318.

FOR THE COMMANDER:

Joseph Karwatka

Encl

JOSEPH KARWATKA
MAJ, MS
Chief, Health and Veterinary Services Office

CF:
Cdr, USAAMCCOM (AMSMC-SG), Rock Island, IL 61299
Cdr, USAEHA (HSHB-MO-T), APG, MD 21010-5423
SMCCR-PPP(Mr. Forcheimer) (w/o encl)

INQUIRE=DOC26D
ITEM NO=00586372
ENVELOPE
CDSN = LGX638 MCN = 94172/00299 TOR = 941720019
OTTUZYUW RUEKJCS3631 1720007-UUUU--RUEALGX.
ZNR UUUUU
HEADER
O 210007Z JUN 94
FM SECDEF WASHINGTON DC
INFO RUEALGX/SAFE
O 202322Z JUN 94
FM SECDEF WASHINGTON DC//OATSD:PA/DPL//
TO RUENAAA/CHINFO WASHINGTON DC
RUEADWD/DA WASHINGTON DC//SAPA//
RUEAHQA/CSAF WASHINGTON DC//SAF/PA//
RUEACMC/CMC WASHINGTON DC//DIVINFO//
INFO RUEKJCS/SECDEF WASHINGTON DC//OATSD:PA//
BT
CONTROLS
UNCLAS SECTION 01 OF 02

/********** THIS IS A COMBINED MESSAGE **********/
BODY
SUBJ: PERSIAN GULF ILLNESSES COMPREHENSIVE CLINICAL EVALUATIONS
1. ON MAY 12, 1994 THE DEPARTMENT OF DEFENSE LAUNCHED A NEW THREE-POINT PLAN TO BETTER UNDERSTAND THE MEDICAL NATURE OF THE PERSIAN GULF SYNDROME. THIS PROGRAM WILL ALLOW THE DEPARTMENT TO BETTER CARE FOR GULF WAR VETERANS WHO ARE ILL WITH NO CLEARLY DEFINED DIAGNOSES. THE THREE-POINT PLAN WILL BE CARRIED OUT IN COORDINATION WITH THE DEPARTMENTS OF VETERANS AFFAIRS AND HEALTH AND HUMAN SERVICES.
2. ASSISTANT SECRETARY OF DEFENSE FOR HEALTH AFFAIRS STEPHEN JOSEPH, MD, DESIGNED THIS PROGRAM BASED ON THE FINDINGS OF THE NATIONAL INSTITUTE OF HEALTH'S TECHNOLOGY WORKSHOP. THE WORKSHOP, HELD APRIL 27-29, 1994, FOUND THAT THE PERSIAN GULF SYNDROME IS NOT A SINGLE DISEASE, BUT RATHER A RANGE OF ILLNESSES WITH OVERLAPPING SYMPTOMS AND CAUSES.
3. THE FIRST OF THE THREE POINTS IS TO CONDUCT A COORDINATED, AGGRESSIVE, COMPREHENSIVE DIAGNOSTIC EFFORT TO DETERMINE AS FAR AS POSSIBLE, THE CAUSES OF THE SYMPTOMS DESCRIBED BY THE NIH WORKSHOP. ALL THOSE PERSIAN GULF VETERANS WHO ARE IN THE DOD'S PERSIAN GULF VETERANS HEALTH SURVEILLANCE SYSTEM WILL BE OFFERED AN INTENSIVE EXAMINATION.
4. TO CONDUCT THE EXAMINATION, A DEFINED, STANDARD PROTOCOL HAS BEEN DEVELOPED BY CLINICAL EXPERTS OF THE MILITARY SERVICES. SENIOR MILITARY PHYSICIANS AND ADMINISTRATORS FROM MILITARY MEDICAL CENTERS CONVENED IN WASHINGTON, DC, ON JUNE 9, 1994, FOR AN ORIENTATION SESSION ON HOW THE CLINICAL EXAMINATIONS WOULD BE CONDUCTED AND ON THE DETAILS OF THE STANDARD PROTOCOL.
5. THE MILITARY PHYSICIANS AND ADMINISTRATORS WILL RETURN TO THEIR MEDICAL CENTERS AND IN TURN INSTRUCT THE MEDICAL AND ADMINISTRATIVE STAFFS IN HOW TO CONDUCT THE COMPREHENSIVE EXAMS.

#567

THERE ARE CURRENTLY ABOUT 300 INDIVIDUALS ON THE DEFENSE ACTIVE DUTY PERSIAN GULF REGISTRY. THESE INDIVIDUALS WILL BE EXAMINED AND THE RESULTS WILL BE REPORTED TO THE DEPUTY SECRETARY OF DEFENSE BY DR. JOSEPH WITHIN 120 DAYS.

6. DURING THE COURSE OF THESE EXAMS, ANY PATIENTS WISHING TO SPEAK TO MEMBERS OF THE MEDIA MAY DO SO AT THEIR OWN ARRANGING. MILITARY PHYSICIANS AND ADMINISTRATORS MAY ALSO SPEAK WITH MEMBERS OF THE MEDIA, IF THEY WISH TO DO SO. WHILE THE HANDLING OF THE EXAMINATIONS WILL BE OPEN FOR REVIEW, THE CONFIDENTIALITY OF ALL PATIENTS WILL BE STRICTLY OBSERVED.

7. AS ADDITIONAL INFORMATION BECOMES AVAILABLE, THE DEPARTMENT WILL PROVIDE PUBLIC COMMENT AND GUIDANCE TO PUBLIC AFFAIRS STAFFS.

8. RETRANSMIT THIS MESSAGE AS APPROPRIATE TO YOUR SUBORDINATE COMMANDS.

9. THE FOLLOWING QUESTIONS AND ANSWERS ARE PROVIDED:

Q1: HOW DO YOU GET ON THE REGISTRY?

A1: ACTIVE DUTY, RETIREES, READY RESERVISTS, AND FULL-TIME NATIONAL GUARDSMEN WHO ARE PERSIAN GULF WAR VETERANS AND THE FAMILY MEMBERS OF ALL THESE INDIVIDUALS WHO ARE ELIGIBLE BENEFICIARIES OF THE DOD HEALTH CARE SYSTEM NEED TO GO TO THEIR MILITARY TREATMENT FACILITY AND ASK TO BE INCLUDED IN THE COMPREHENSIVE CLINICAL EVALUATION PROGRAM. OTHER PERSIAN GULF WAR VETERANS AND THEIR FAMILY MEMBERS SHOULD CONTACT THEIR CLOSEST DEPARTMENT OF VETERANS AFFAIRS HOSPITAL OR CALL THE FOLLOWING TOLL FREE NUMBER 1-800-796-9699, WHICH WILL BE ACTIVATED ON JUNE 23D AT NOON EDT.

Q2: WHO IS ELIGIBLE TO BE ON THE REGISTRY?

A2: ACTIVE DUTY, RETIREES, READY RESERVISTS, FULL-TIME NATIONAL GUARD, FAMILY MEMBERS WHO ARE ELIGIBLE BENEFICIARIES.

Q3: WHERE DOES ONE GO FOR THE COMPREHENSIVE CLINICAL EVALUATION PROGRAM (CCEP)?

A3: THE FIRST STAGE OF THE CLINICAL EVALUATION IS TO BE SEEN AT THE LOCAL MILITARY TREATMENT FACILITY. IF THE EVALUATION IS INCONCLUSIVE OR IF THE PATIENT IS NOT SATISFIED THAT THE DIAGNOSIS ADEQUATELY EXPLAINS THEIR HEALTH PROBLEM, THE PATIENT WILL BE REFERRED TO ONE OF THE 16 MILITARY REGIONAL MEDICAL CENTERS WHERE ADDITIONAL EVALUATIONS WILL OCCUR. IF A DIAGNOSIS STILL CANNOT BE REACHED, THE RECORDS OF ALL THE EVALUATIONS WILL BE REVIEWED BY A SPECIAL CLINICAL REVIEW COMMITTEE THAT INCLUDES INDIVIDUALS FROM THE INSTITUTE OF MEDICINE OF THE NATIONAL ACADEMY OF SCIENCES IN WASHINGTON, DC.

Q4: WHAT SHOULD ONE EXPECT FROM THE CCEP?

A4: THIS IS A METHODICAL APPROACH TO PROVIDING MEDICAL CARE TO INDIVIDUALS WHO FEEL THAT THEY HAVE AN ILLNESS AS A RESULT OF SOME ASSOCIATION WITH THE PERSIAN GULF WAR. INDIVIDUALS WHO PARTICIPATE IN THE COMPREHENSIVE CLINICAL EVALUATION PROGRAM CAN EXPECT TO RECEIVE COMPETENT, TIMELY AND PROFESSIONAL MEDICAL CARE.

Q5: HOW LONG WILL THE CCEP PROCESS TAKE? WILL PEOPLE STAY OVERNIGHT?

A5: THE TIME REQUIRED FOR THIS EVALUATION WILL VARY WITH EACH INDIVIDUAL AND THE TYPE OF PROBLEM THEY ARE EXPERIENCING. PATIENTS WILL BE ADMITTED TO THE HOSPITAL AS NECESSARY TO ENSURE THAT THE EVALUATIONS ARE COMPLETED.

Q6: DOES THE INDIVIDUAL'S COMMAND CUT ORDERS FOR TRAVEL/PER DIEM?

A6: ORDERS FOR TRAVEL WILL BE ISSUED BY THE COMMAND OR THE MILITARY TREATMENT FACILITY IN ACCORDANCE WITH CURRENT LAWS AND REGULATIONS.
Q7: TO WHOM WOULD A PERSON REPORT?
A7: INDIVIDUALS WHO ARE BEING REFERRED TO A REGIONAL MEDICAL CENTER FOR FURTHER EVALUATION WILL BE GIVEN SPECIFIC INSTRUCTIONS BY THE MILITARY TREATMENT FACILITY FROM WHICH THEY ARE BEING REFERRED.
Q8: DOES THE HOSPITAL KNOW THE PERSON IS ENROUTE?
A8: YES. THE MILITARY TREATMENT FACILITY THAT REFERS THE PATIENT WILL BE IN CONTACT WITH THE INDIVIDUALS RESPONSIBLE FOR THE COMPREHENSIVE CLINICAL EVALUATION PROGRAM AT THE REGIONAL MEDICAL CENTER TO WHICH THEY ARE REFERRED.
Q9: HOW MANY HOSPITALS? WHERE?
A9: THERE ARE FOURTEEN HOSPITALS IN CONUS AND TWO OCONUS. THESE HOSPITALS INCLUDE THE TWELVE REGIONAL MEDICAL CENTERS PLUS OTHER MAJOR MEDICAL CENTERS IN THOSE REGIONS AND ONE IN EUROPE.
Q10: WHEN WILL THOSE EVALUATED KNOW THE RESULTS?
A10: THE RESULTS OF THE INDIVIDUAL EVALUATIONS WILL BE COMMUNICATED JUST LIKE ANY OTHER VISIT TO THE DOCTOR OR ADMISSION TO THE HOSPITAL. THE PRIMARY GOAL OF THIS PROGRAM IS TO MAKE SURE PEOPLE RECEIVE THE HEALTH CARE THEY NEED.
Q11: WHAT HAPPENS IF TREATMENT IS REQUIRED?
A11: TREATMENT WILL BE ADMINISTERED AS REQUIRED AND AS THE PHYSICIAN FEELS IS NECESSARY.
Q12: WHAT DOES A PERSON STATIONED IN EUROPE OR THE PACIFIC DO?
A12: THEY WOULD GO TO A MEDICAL TREATMENT FACILITY AND FOLLOW THE SAME PROCEDURES AS IN THE CONTINENTAL UNITED STATES. THE DESIGNATED CENTERS ARE LANDSTUHL ARMY REGIONAL MEDICAL CENTER IN GERMANY AND TRIPLER ARMY MEDICAL CENTER FOR THE PACIFIC AREA.
Q13: WILL THE EVALUATIONS BE THE SAME FROM HOSPITAL TO HOSPITAL? IN ALL SERVICES? AS IN THE DVA?
A13: THE MINIMAL EVALUATIONS TO BE CONDUCTED IN EACH PHASE OF THIS CLINICAL PROTOCOL HAVE BEEN STANDARDIZED ACROSS THE DOD HOSPITALS.

/****** BEGINNING OF SECTION 002 ******/
THIS PROTOCOL HAS BEEN DEVELOPED IN CONJUNCTION WITH THE DEPARTMENT OF VETERANS AFFAIRS TO ENSURE CONSISTENCY BETWEEN THE TWO DEPARTMENTS.
Q14: CAN PEOPLE CHOOSE WHERE THEY GO FOR AN EVALUATION--MIL, OR MIL/VA?
A14: INDIVIDUALS SHOULD GO TO THEIR CLOSEST MILITARY TREATMENT FACILITY INITIALLY. IF THEY NEED TO BE REFERRED TO A REGIONAL MEDICAL CENTER, THEY WILL ORDINARILY BE REFERRED TO THE MEDICAL CENTER FOR THEIR REGION.
Q15: HOW DO FAMILY MEMBERS GET EVALUATED?
A15: FAMILY MEMBERS WHO ARE ELIGIBLE BENEFICIARIES GO TO A MEDICAL TREATMENT FACILITY AND FOLLOW THE SAME PROCEDURES AS PREVIOUSLY OUTLINED.
Q16: HOW DO PEOPLE KNOW THEY RECEIVED THE WHOLE EVALUATION?
A16: EACH INDIVIDUAL WHO PARTICIPATES IN THE COMPREHENSIVE CLINICAL EVALUATION PROGRAM WILL BE GIVEN A "PATIENTS BILL OF RIGHTS" THAT DESCRIBES THE ENTIRE PROGRAM. SPECIFIC PROCEDURES WILL BE DISCUSSED

INQUIRE=DOC21D
ITEM NO=00259602
ENVELOPE
CDSN = LGX546 MCN = 93078/09609 TOR = 930780621
RTTUZYUW RUEKJCS1278 0780618-UUUU--RUEALGX.
ZNR UUUUU
HEADER
R 190618Z MAR 93
FM DIA WASHINGTON DC
INFO RUEALGX/SAFE
R 182231Z MAR 93
FM SECDEF WASHINGTON DC//OSD:PA//
TO AIG 8798
AIG 8799
BT
CONTROLS
UNCLAS SECTION 01 OF 04
FOR PUBLIC AFFAIRS OFFICERS

/********** THIS IS A COMBINED MESSAGE **********/
BODY
SUBJ: DOD NEWS BRIEFING
DELIVER DURING NORMAL DUTY HOURS
FOLLOWING IS TRANSCRIPT OF NEWS BRIEFING CONDUCTED BY MR. BOB HALL, DEPUTY ASSISTANT SECRETARY OF DEFENSE/PUBLIC AFFAIRS, AT THE PENTAGON, ON THURSDAY, MARCH 18, 1993, AT 1:00 P.M.:
MR. HALL: GOOD AFTERNOON.
GENERAL COLIN L. POWELL, CHAIRMAN OF THE JOINT CHIEFS OF STAFF, WILL SPEAK AT THE COMSTOCK CLUB IN SACRAMENTO, CALIFORNIA, ON FRIDAY, MARCH 19TH. THE TOPIC OF HIS SPEECH WILL BE: "FACTS ABOUT AMERICA'S DEFENSE.: THE NOON EVENT WILL BE HELD AT THE RED LION BALL ROOM AT THE RED LION HOTEL. MEDIA WHO DESIRE TO COVER THE EVENT SHOULD CONTACT MS. JOAN ASBOTH IN THE CHAIRMAN'S PUBLIC AFFAIRS OFFICE AT (703) 697-4272. OR MR. DON JOHNSON, THE EXECUTIVE SECRETARY FOR THE COMSTOCK CLUB, AT (916) 442-4608.
SECOND, YOU MAY HAVE NOTICED IN THE RECENT RELEASES THAT WE HAVE BEEN REFERRING TO THE DEFENSE ADVANCE RESEARCH PROJECT AGENCY, BETTER KNOWN AS DARPA, AS THE ADVANCED RESEARCH PROJECT AGENCY, OR ARPA. OBVIOUSLY, WE'RE DROPPING THE D FOR DEFENSE. THIS REFLECTS THE PRESIDENT'S INITIATIVE THAT HE ANNOUNCED LAST WEEK. WE HAVE THE OFFICIAL PAPERWORK ON THE CHANGE OF THE TITLE. DEPUTY SECRETARY OF DEFENSE WILLIAM J. PERRY SIGNED THE MEMO DIRECTING THE CHANGE ON MARCH 15TH FOR THE OFFICIAL RECORD.
THAT'S ALL I HAVE FOR ANNOUNCEMENTS.
Q: THE STATE DEPARTMENT REPORTS THAT THERE WAS AN INCURSION BY IRANIAN JETS IN THE NORTHERN NO-FLY ZONE OF IRAQ TWO DAYS AGO. CAN YOU TELL US THE NUMBER OF PLANES INVOLVED, THE TYPE OF PLANES? DOES THE UNITED STATES AND ITS COALITION ALLIES PLAN TO ENFORCE THE NO-FLY ZONE BY SHOOTING DOWN, INCLUDING IRANIAN PLANES COMING IN? AND IS THERE ANY PLANNED RETALIATION BY THE UNITED STATES FOR THE BOMBING OF THE KURDS?

A: BASICALLY, I'VE GOT NOTHING BEYOND WHAT THE STATE DEPARTMENT SAID ON TUESDAY -- THAT IRANIAN AIRCRAFT ATTACKED IRANIAN KURDISH DISSIDENTS INSIDE THE NO-FLY ZONE NORTH OF THE 36TH PARALLEL IN NORTHERN IRAQ. THAT ATTACK OCCURRED OVER THE WEEKEND. OUR POLICY CONTINUES TO BE TO SUPPORT THE TERRITORIAL INTEGRITY, UNITY, AND SOVEREIGNTY OF IRAQ, AND NO FLIGHTS OF ANY KIND OTHER THAN COALITION AIRCRAFT ARE PERMITTED NORTH OF THE 36TH PARALLEL OR SOUTH OF THE 32ND PARALLEL, AND IRAN IS WELL AWARE OF THE POLICY.

Q: WHY WASN'T ACTION TAKEN AGAINST THE IRANIAN PLANES? CERTAINLY, IF IRAQI PLANES HAD MOVED INTO THAT AREA, THEY WOULD HAVE BEEN BLOWN OUT OF THE SKY.

A: THE ISSUE IS WHETHER WE ARE IN THE AREA AND IN POSITION TO DO SOMETHING ABOUT THE INCURSION. THAT GOES TOWARDS WHOEVER IS MAKING THE INCURSION.

Q: WERE WE AWARE OF IT AT THE TIME IT OCCURRED, OR WAS IT SOME, THING LEARNED AFTER THE FACT?

A: WE WERE NOT AWARE OF IT AT THE TIME IT OCCURRED.

Q: CAN YOU RESTATE THE POLICY, OR GIVE US WHATEVER THE CURRENT PHILOSOPHY IS? IF YOU SAY, AS YOU HAVE IN THE PAST, THAT THE U.S. WILL ENFORCE A NO-FLY ZONE, DOES THIS MEAN THAT IF THERE'S A REPEAT OFFENSE BY ANY AIRCRAFT, INCLUDING IRANIAN, THEY WILL BE SHOT DOWN?

A: I'M NOT GOING TO SPECULATE ABOUT FUTURE ACTION OR RULES OF ENGAGEMENT. I'LL JUST REPEAT, THAT THE NO-FLY ZONE MEANS THAT PLANES SHOULD NOT FLY. I THINK WE TALKED ABOUT THAT, CERTAINLY AT THE TIME THAT WE ANNOUNCED THE SOUTHERN NO-FLY ZONE, AND PROBABLY DURING THE VARIOUS BRIEFINGS ON THE NORTHERN NO-FLY ZONE.

Q: WHAT WAS IRAN TOLD ABOUT THE INCIDENT?

A: I'M SURE THE STATE DEPARTMENT HAS BEEN IN CONTACT THROUGH THE VARIOUS MEANS THAT WE HAVE IN DISCUSSING THINGS WITH IRAN, BUT I'LL LEAVE IT TO THEM.

Q: THE OFFICE OF THE UN HIGH COMMISSIONER FOR REFUGEES HAS ISSUED AN APPEAL FOR EMERGENCY HELP FOR SOME 60,000 INDIVIDUALS THAT ARE TRAPPED AROUND SREBRENICA. THEY'RE SAYING THEY ARE IN DIRE NEED OF FOOD AND BASICALLY OF MEDICAL HELP OR EVEN TRANSIT OUT. THEY'RE ASKING FOR THE AIRDROPS TO BE DONE DURING THE DAY, AND POSSIBLE HELICOPTER AIRLIFTS TO BRING THE WOUNDED OUT. IS THERE A CHANGE IN THE U.S. AIRDROPS THAT'S BEING CONSIDERED TO HELP THESE PEOPLE?

A: I DON'T HAVE ANYTHING TO ANNOUNCE FOR YOU. I'M SURE WE'RE LOOKING AT THE ISSUE OF THE SITUATION IN THE POCKET. BUT I WOULD NOT PROJECT FOR YOU A CHANGE IN OUR METHOD OF OPERATION. AS YOU KNOW, WHEN WE STARTED THESE AIRDROPS, WE TALKED ABOUT THE QUESTION OF BALANCING EFFICIENCY, ACCURACY, WITH THE SAFETY OF OUR CREWS. THE SAFETY OF OUR CREWS CONTINUES TO BE VERY IMPORTANT TO US. SO I THINK THAT WILL BE AMONG THE GOVERNING FACTORS AS WE LOOK AT THE SITUATION.

Q: SO YOU'RE RULING OUT DAYTIME AIRDROPS?

A: I DON'T WANT TO RULE ANYTHING IN OR OUT. BUT I'M JUST NOT PROJECTING ANYTHING FOR YOU. I WANT TO REMIND YOU OF OUR CONCERN ABOUT THE SAFETY OF OUR CREWS. IT WOULD NOT HELP THE SITUATION TO HAVE A U.S. AIRCREW SHOT DOWN.

Q: CAN YOU GIVE US AN UPDATE OF ABOUT HOW MANY DROPS HAVE BEEN

MADE RECENTLY?
A: SURE. LAST NIGHT WAS THE 18TH AIRDROP MISSION TO PROVIDE PROMISE. SINCE WE BEGAN ON MARCH 1ST, WE HAVE DROPPED 544.6 TONS OF FOOD AND 19.3 TONS OF MEDICAL SUPPLIES. IN ADDITION, WE HAVE CONTINUED THE FLIGHTS INTO SARAJEVO. SINCE JULY, THAT HAS TOTALED 861 FLIGHTS WHICH HAVE DELIVERED 10,133 TONS OF RELIEF SUPPLIES AND OTHER CARGO.
LAST NIGHT'S MISSION CONSISTED OF FIVE C-130 CARGO PLANES DROPPING 30.2 TONS OF MATERIAL. THAT INCLUDED 42 BUNDLES OF MILITARY RATIONS WEIGHING 29.1 TONS, AND FIVE BUNDLES OF MEDICAL SUPPLIES WEIGHING 1.1 TON. THE DROPS WERE NEAR SREBRENICA AND GABELA IN EASTERN BOSNIA. THIS IS THE SECOND DROP THERE. WE ALSO DROPPED IN GABELA ON MARCH 16TH.
THE SUPPLIES THAT WE HAVE DROPPED SO FAR HAVE BEEN CONTRIBUTED BY THE UNITED STATES, TURKEY, NORWAY, GREAT BRITAIN, AND THE WORLD HEALTH ORGANIZATION. THEY HAVE CONTRIBUTED OVER 200 POUNDS OF PENICILLIN, WHICH IS AN ITEM OF PARTICULAR NEED. IN THE NEXT COMING DAYS, I WOULD EXPECT TO SEE US ALSO DROPPING SOME UNICEF SUPPLIES AND SOME SUPPLIES THAT ARE BEING PROVIDED BY THE EUROPEAN COMMUNITY.
Q: STAYING WITH EASTERN BOSNIA, AND AS WE TALK ABOUT NO-FLY ZONES, APPARENTLY THERE WAS A VIOLATION OF THIS NO-FLY ZONE OVER THE WEEKEND WHEN SERB PLANES BOMBED MUSLIM VILLAGES IN EASTERN BOSNIA. WHAT'S BEING DONE ABOUT THAT?
A: BASICALLY, WE HAVE THE UNITED NATIONS REPORT. I DON'T HAVE ANYTHING ADDITIONAL TO ADD TO THAT. AS YOU KNOW, WE'VE BEEN DISCUSSING FOR SOME TIME THE QUESTION OF ENFORCEMENT OF THE NO-FLY ZONE, BUT THERE HAS BEEN NO UN RESOLUTION TO PUT THAT INTO EFFECT.
Q: DOES IT CAUSE CONCERN, HOWEVER, WHEN YOU'RE DOING AIRDROPS OVER THE SAME AREA?
A: ONE OF THE REASONS WHY WE WANTED A NO-FLY ZONE, IN ADDITION TO PREVENTING THE USE OF AIRCRAFT TO CONDUCT MILITARY STRIKES ON

/****** BEGINNING OF SECTION 002 ******/
INNOCENT PEOPLE, AND TO REDUCE THE LEVEL OF FIGHTING, IS TO AVOID ANY INTERFERENCE WITH THE UN RELIEF FLIGHTS, OR THE UN,SPONSORED RELIEF FLIGHTS INTO SARAJEVO AND ALSO OUR AIRDROP OPERATIONS. THE NO-FLY ZONE ISSUE CAME UP BEFORE WE STARTED DOING THE AIRDROPS, SO IT REALLY HAD TO DO WITH THE TOTAL UN OPERATIONS.
Q: THE BOSNIAN AMBASSADOR SAID THERE WERE SOME VIOLATIONS AGAIN LAST NIGHT. I THINK YOU WERE REFERRING TO THE ONES THAT TOOK PLACE OVER THE WEEKEND. DO YOU HAVE ANYTHING AT ALL ON THE ONE LAST NIGHT?
A: THESE MAY HAVE BEEN. THERE HAVE BEEN A NUMBER OF VIOLATIONS OF THE NO-FLY ZONE.
Q: THIS INVOLVING BOMBING?
A: I JUST DON'T HAVE ANY NEW INFORMATION. AGAIN, THAT WAS A UN REPORT. I'LL SOURCE YOU BACK TO THE UNITED NATIONS FOR THAT.
LET ME JUST GO ON TO SAY, IN ANSWER TO SUSANNE'S QUESTION, WE ARE CONCERNED ABOUT THE SITUATION IN SREBRENICA. WE ESTIMATE THAT THERE ARE UPWARDS OF 50,000 REFUGEES WHO HAVE BEEN DRIVEN FROM

THEIR HOMES IN OTHER AREAS WHO HAVE MOVED INTO THIS POCKET. WE KNOW THAT IT'S A VERY DESPERATE SITUATION FOR MANY OF THEM. WE KNOW THAT THE AIRDROPS CANNOT SUPPLY ALL THEIR NEEDS. BUT THAT'S NOT NEW INFORMATION. THAT'S SOMETHING WE'VE ALWAYS KNOWN. BUT IT DOESN'T MAKE IT ANY LESS NECESSARY TO CONTINUE TO DROP THE SUP PLIES. WE KNOW FROM EYE WITNESSES, INCLUDING UNPROFOR OFFICIALS, UNHCR, AND WORLD HEALTH ORGANIZATION PERSONNEL ON THE GROUND, AS WELL AS JOURNALISTS, THAT THE SUPPLIES ARE REACHING PEOPLE THAT NEED THEM. BUT WE ALSO NOTE THAT THE DISTRIBUTION SYSTEM IN NOT ADEQUATE TO MEET THE NEEDS OF ALL THE PEOPLE -- JUST THE SHEER NUMBERS OF REFUGEES AS WELL AS THE CHAOS OF THE SITUATION. IT'S SOMETHING THAT WE'RE WORKING ON ON A VARIETY OF FRONTS INTERNATION, ALLY.

Q: HASN'T IT BEEN STATED BEFORE THAT THERE'S NO WAY THE AIRDROPS COULD SUPPLY ALL THE NEEDS, THAT ULTIMATELY YOU'RE TRYING TO GET THE LAND CONVOYS MOVING? WHERE DOES THAT...

A: IN TERMS OF SUPPLYING HUMANITARIAN RELIEF, THAT IS THE RIGHT SOLUTION -- TO GET THE LAND CONVOYS IN. OF COURSE, THE OVERALL SOLUTION IS TO GET A PEACE AGREEMENT.

Q: ARE WE SEEING ANY IMPROVEMENT IN THE MOVEMENT OF LAND CONVOYS?

A: I HAVEN'T DONE A CHECK ON THAT IN THE LAST COUPLE OF DAYS. AS YOU KNOW, I'VE BEEN OUT WITH THE FLU. BUT I'LL CHECK FOR YOU. WE HAVE SOME PRESS REPORTS THAT SAY THAT A NUMBER HAVE GOTTEN THROUGH TO OTHER AREAS, ZEPA AND TUZLA, BUT I JUST DON'T... WE'LL TRY GO GET OUR OWN INFORMATION FOR YOU.

Q: ARE THESE SIGNIFICANT? ARE THEY MAKING A DIFFERENCE?

A: WE'LL TAKE THAT.

Q: KOREA. HAS THE SOUTH KOREAN GOVERNMENT ASKED OUR FORCES TO STAY ON AFTER TEAM SPIRIT? AND WILL THE U.S. FORCES THAT DEPART FROM TEAM SPIRIT BE STAYING ON IN SOUTH KOREA AFTER TDDAY?

A: NO, AND NO. NO, OUR FORCES HAVE NOT BEEN ASKED TO STAY ON; AND NO, THEY WILL NOT STAY ON. IN FACT, THE EXERCISE PORTION OF TEAM SPIRIT ENDS TODAY. THERE WILL BE A PRESS CONFERENCE OUT THERE IN KOREA AT THE MINISTRY OF NATIONAL DEFENSE PRESS ROOM IN SEOUL AT 10:00 A.M. TOMORROW, LOCAL TIME, IN KOREA.

THE DEPLOYMENT PHASE OF THE EXERCISE BEGAN IN JANUARY. THE FIELD TRAINING PORTION BEGAN ON MARCH 9TH. AS I SAID, THE FIELD TRAINING PORTION OF THE EXERCISE ENDED TODAY. THE REDEPLOYMENT OF THE AUGMENTING FORCES WILL CONTINUE THROUGH MID,APRIL. BUT ALL OF OUR FORCES THAT WE SENT THESE FOR THE EXERCISE WILL LEAVE.

Q: NORTH KOREA IS ALSO PUTTING PRESSURE ON SOUTH KOREA TO HAVE THEM ASK THAT ALL U.S. FORCES LEAVE THE COUNTRY. WHAT'S THE DEPARTMENT'S POSITION ON THAT?

A: WE HAVE A CLOSE BILATERAL RELATIONSHIP WITH THE REPUBLIC OF KOREA. I SUSPECT THAT IT WILL CONTINUE. OBVIOUSLY, WE'RE THERE AT THEIR INVITATION, IN RESPONSE TO THE SECURITY SITUATION THAT THEY FACE.

Q: A WATCHDOG GROUP IS CLAIMING THAT THE USE OF, I GUESS IT'S THE DEPLETED URANIUM PRDJECTILES IN DESERT STORM, IS CAUSING THE MYSTERIOUS ILLNESS OF U.S. FORCES. ANY REACTION?

A: I'M NOT AWARE OF ANY EVIDENCE THAT ILLNESSES HAVE BEEN CAUSED

BY THE USE OF DEPLETED URANIUM. HOWEVER, WE'VE GOT A LOT OF INFORMATION ON THAT BACK IN DDI. COLONEL MEEHAN CAN GO THROUGH ALL THE HISTORY OF THIS WITH YOU, THE VARIOUS STUDIES THAT HAVE BEEN DONE AND ARE BEING DONE TO KEEP AN EYE ON THE SITUATION, TO MAKE SURE THAT WE WON'T HAVE SOME PROBLEM THAT HAS DEVELOPED. I'M NOT AWARE OF ANY PROBLEM.

Q: THE STARS & STRIPES HAS HAD A REPORT THAT MOST WOMEN IN THE MILITARY IN EUROPE CAN'T GET ABORTIONS, DESPITE SECRETARY ASPIN'S DIRECTIVE TO LIFT THE BAN. THEY ALSO CITE THAT IT SEEMS TO BE THE ABILITY OF THE HEALTH CARE PROFESSIONALS INVOLVED TO OPT OUT OF SUCH PROCEDURES. IS THERE ANY EFFORT BEING MADE TO BRING IN PHYSICIANS AND NURSES WHO ARE WILLING TO PROVIDE THOSE PROCEDURES?

A: LET ME WALK THROUGH A LITTLE BIT OF THE HISTORY. IN 1982, CONGRESS PASSED WHAT WAS CALLED THE HYDE AMENDMENT, THAT PROHIBITED THE FUNDING FOR ABORTIONS IN DOD FACILITIES UNLESS THE LIFE OF THE MOTHER WAS IN DANGER. DOD ADHERED TO THE LEGISLATION AND DID NOT USE TAXPAYER FUNDS TO PAY FOR ELECTIVE ABORTIONS. HOWEVER, WE DID ALLOW BENEFICIARIES TO PAY FOR THEIR ABORTIONS IN MILITARY FACILI, TIES OVERSEAS, WHERE QUALITY CARE WAS NOT LOCALLY AVAILABLE. THAT WAS THE GOVERNING POLICY -- OVERSEAS WHERE QUALITY CARE WAS NOT LOCALLY AVAILABLE. THIS PRACTICE WAS KNOWN AS PREPAID ABORTIONS. IN 1988, WE REVIEWED THE POLICY OF PREPAID ABORTIONS, AND ALTHOUGH NO LEGAL VIOLATIONS WERE FOUND, A DETERMINATION WAS MADE THAT THE PRACTICE SHOWED INSENSITIVITY TO THE SPIRIT OF THE CONGRESSIONALLY ENACTED POLICY OF WITHHOLDING GOVERNMENT INVOLVEMENT IN THE PROVI-SION OF ABORTIONS.

CONSEQUENTLY, ALL PREPAID ABORTIONS IN MILITARY TREATMENT FACILI, TIES OVERSEAS WERE STOPPED AS OF OCTOBER 1, 1988. THERE WAS A MEMO THAT DIRECTED THAT.

DURING THAT PERIOD OF '82 TO '88, I CAN GIVE YOU THE FIGURES. THE SERVICES PERFORMED 100 PREPAID ABORTIONS OVERSEAS IN 1983; 37 IN 1984; 12 IN 1985; 17 IN 1986; FIVE IN 1987; AND EIGHT IN 198. ON JANUARY 22ND OF THIS YEAR, PRESIDENT CLINTON REVERSED THE 1988 BAN ON PREPAID ABORTIONS IN OVERSEAS HOSPITALS. CONSEQUENTLY, SECRE TARY ASPIN SENT IMPLEMENTING INSTRUCTIONS TO THE MILITARY SERVICES ON FEBRUARY 4, 1993. IN THAT, HE DIRECTED THEM TO REINSTATE THE ABORTION POLICY PRIOR TO OCTOBER 1, 1988. SO IT WENT BACK TO THE POLICY OF ALLOWING PREPAID ABORTIONS IN U.S. OVERSEAS FACILITIES. THE CHARGES FOR THOSE ABORTIONS WERE PLACE AT THE EXISTING RATE FOR SAME-DAY SURGERY WHICH IN FY93 IS $477 A DAY.

THE SECRETARY ALSO STATED THAT OTHER RELEVANT DOD POLICIES AND PRACTICES APPLY. IN PARTICULAR, HOST NATION PROHIBITIONS ON SUCH PROCEDURES MUST BE FOLLOWED. AND IT HAS BEEN A PRACTICE OF THE MILITARY SERVICES THAT MEDICAL PERSONNEL WILL NOT BE FORCED TO ACT IN CONFLICT WITH THEIR OWN MORAL, ETHICAL OR RELIGIOUS CONVICTIONS FOR ELECTIVE PROCEDURES. THAT PRACTICE IS CONTINUING.

/******* BEGINNING OF SECTION 003 *******/

THE MILITARY DEPARTMENTS ARE CURRENTLY DEVELOPING THE IMPLEMENTING INSTRUCTIONS AS A RESULT OF THE SECRETARY'S MEMO. IN EUROPE, FOR EXAMPLE, THE ARMY IS SURVEYING MEDICAL PERSONNEL,

TALKING TO THEM ABOUT THOSE WHO HAVE CONFLICTS WITH THE POLICY, IDENTIFYING HOST NATION LAWS TO SEE IF WE HAVE ANY CONFLICTS WITH LAWS OF THE HOST NATIONS, AND DEVELOPING A CONCEPT ON HOW THE COMMAND WILL COMPLY WITH THE PRESIDENT'S ORDER.
ALTHOUGH SOME PHYSICIANS HAVE EXPRESSED UNWILLINGNESS TO PERFORM THE PROCEDURE, THE MILITARY SERVICES WILL IMPLEMENT THE POLICY, AND WITH THE POSSIBILITY THAT GYNECOLOGISTS WILL BE AVAILABLE IN CENTRAL LOCATIONS TO PERFORM ABORTIONS. IN ADDITION, I WOULD NOTE THAT WHILE THESE POLICIES ARE BEING FINALIZED, ABORTION SERVICES ARE LEGALLY AVAILABLE IN MOST EUROPEAN HOST NATION CIVILIAN FACILITIES AT NO GREATER FEE THAN IS BEING CHARGED BY THE MILITARY FACILITIES.

IN SUM, THEY'RE WORKING TO DEVELOP AN EFFECTIVE, QUALITY MEDICAL PRACTICE THAT IMPLEMENTS THE NATIONAL POLICY. DURING THIS TIME, REQUESTS FOR ABORTIONS ARE BEING CONSIDERED. IN FACT, THE ARMY IN EUROPE REPORTS THAT IT HAS RECEIVED ONLY ONE ABORTION REQUEST SINCE THE POLICY WAS ANNOUNCED, AND THAT WOMAN IS BEING ACCOMMODATED.

Q: SINCE THE MORAL BELIEFS OF THESE INDIVIDUALS ARE BEING TAKEN INTO ACCOUNT IN REGARDS TO MEDICAL PROCEDURES, WHAT ABOUT THE MORAL BELIEFS THAT SERVICE PEOPLE HAVE IN REGARD TO HOMOSEXUALITY IN THE SERVICES?

A: ONE IS A QUESTION OF A STANDARD PRACTICE FOR DOCTORS. YOU DON'T ASK THEM TO PERFORM ELECTIVE OPERATIONS WHEN IT CONFLICTS WITH THEIR MORAL BELIEFS. THAT WOULD BE OVERRIDDEN IF THE LIFE OF THE PERSON WAS IN DANGER. I'M NOT SURE I SEE THE CONNECTION TO THE OTHER POLICY QUESTION.

Q: I'M WONDERING, ARE THEY TAKING INTO CONSIDERATION THE FACT THAT PEOPLE DON'T AGREE WITH THE OPEN PRACTICE OF HOMOSEXUALITY, SHOULD THEY THEN BE FORCED TO SERVE WITH --

A: WITHIN THE PURVIEW OF THE REVIEW THAT IS BEING DONE IN THE DEPARTMENT OF DEFENSE ABOUT HOW TO IMPLEMENT THE PRESIDENT'S DECISION, THAT'S CERTAINLY ONE OF THE ISSUES THAT WOULD COME INTO PLAY.

Q: CAN I GO BACK TO IRAN/IRAQ FOR JUST A SECOND? WOULD YOU TAKE THE QUESTION TO FIND OUT, IF POSSIBLE, HOW MANY AIRCRAFT WERE INVOLVED, TYPE OF AIRCRAFT, TYPE OF ORDNANCE DROPPED, DAMAGE, INJURIES, ANYTHING ELSE ABOUT THE RAID 48 HOURS AGO?

A: I DON'T BELIEVE WE HAVE INDEPENDENT INFORMATION. THE REPORTS COME FROM THE IRANIANS THEMSELVES, THE KURDS ON THE SCENE, AND I BELIEVE THE FRENCH.

Q: IS THE PENTAGON FUNCTIONING AS IF LES ASPIN IS STILL IN CHARGE, EVEN THOUGH HE'S WIRED FOR SOUND OVER IN GEORGETOWN?

A: THE SECRETARY IS, OF COURSE, STILL IN CHARGE. HE STILL HAS COMMUNICATIONS. HE'S BEEN MEETING WITH HIS STAFF MEMBERS ON A REGULAR BASIS WHILE HE'S BEEN IN THE HOSPITAL. AND, OF COURSE, WE NOW HAVE A SLIGHTLY DIFFERENT AND BETTER SITUATION, WE DO HAVE THE DEPUTY SECRETARY HERE IN PLACE, CONFIRMED, SWORN IN, ABLE TO ACT IF NECESSARY.

Q: HAS THIS AFFECTED THE BUDGET SCHEDULE, TIME LINES, DOCUMENTS THAT NEED TO GET OVER TO OMB, ET CETERA? HAVE THOSE CHANGED?

A: NOT SO FAR. OBVIOUSLY, THE SECRETARY HAD BEEN PLANNING TO GO TESTIFY ON THE HILL. HE HAD POSTPONED THAT BECAUSE HE BASICALLY SAID THE MATERIALS HE WANTED TO USE WEREN'T READY. BUT THAT WAS GOING TO BE POSTPONED AND BE RESCHEDULED THIS WEEK. THAT HASN'T HAPPENED BECAUSE HE'S IN THE HOSPITAL.

Q: THE NEW BUDGET HAS BEEN SUBMITTED TO OMB. IT JUST HAS NOT COME BUT IN BLESSED FORMULA?

A: YES, THAT'S TRUE. LET ME TAKE THE QUESTION, HAVE WE SENT THE TAPES OVER TO OMB?

Q: AND WHAT DAY WERE THEY SENT?

A: OKAY.

Q: HAS THE SECRETARY SPOKEN WITH GENERAL POWELL OR IS THAT SOMETHING THAT IS BEING PASSED ON TO MR. PERRY?

A: I DON'T KNOW IF HE SPOKE TO HIM TODAY.

Q: I'M MUST WONDERING WHAT TYPE OF WORK THE SECRETARY MIGHT BE CONDUCTING, GIVEN HIS SITUATION, OBVIOUSLY.

A: I, FRANKLY, DON'T KNOW WHAT HE DID TODAY. TODAY HE HAD THE OPERATION. YOU ALL GOT BRIEFED ON THAT OVER AT THE HOSPITAL' BUT I KNOW, SINCE HE'S BEEN IN THE HOSPITAL, HE'S HAD REGULAR MEETINGS WITH PEOPLE' AND GENERAL POWELL, I KNOW, HAS IN THE PAST PARTICIPATED IN THOSE. WHETHER HE DID THIS WEEK OR NOT, I'M NOT SURE.

Q: CAN YOU TAKE THE QUESTION?

A: HAS HE MET WITH GENERAL POWELL THIS WEEK?

Q: WELL, IF HE DID ANY WORK TODAY AT ALL, SIGNED A PAPER OR ANYTHING' I JUST WANT TO GET THAT CLARIFIED. IF NOT, THAT'S FINE, BUT I JUST WANT TO KNOW ONE WAY OR ANOTHER.

Q: DO YOU HAVE WHAT HIS CONDITION IS NOW? IS IT SATISFACTORY?

A: NOTHING NEW SINCE YOU ALL GOT THE PRESS CONFERENCE WITH THE DOCTORS OVER AT...

Q: ...THIS PROCEDURE, I ASSUME?

A: YES, THERE WAS A PRESS CONFERENCE POST,PROCEDURE. YOU ALL HEARD IT. IF NOT, WE'LL GET THE TRANSCRIPT FOR YOU. I HAVEN'T HAD A CHANCE TO HEAR OR READ IT.

Q: WILL WE GET AN UPDATE TOMORROW, SINCE THERE IS NO BRIEFING, ON HIS HEALTH PLAN FOR OVER THE WEEKEND, AND SOME KIND OF PROGNOSIS, WHEN TO EXPECT HIS DEPARTURE FROM THE HOSPITAL?

A: OKAY'

Q: KISMAYO. WHAT'S THE SITUATION? WE HAD REPORTS EARLIER THIS WEEK OF FACTIONAL FIGHTING BETWEEN THE WARLORDS, AND THAT U.S. TROOPS COULD BECOME INVOLVED.

A: IT'S CURRENTLY QUIET IN KISMAYO. NO RECENT INCIDENTS. A REACTION FORCE OF APPROXIMATELY 500 U.S. ARMY TROOPS IS NOW IN PLACE IN KISMAYO. THEY'VE BEEN SENT TO AUGMENT THE BELGIANS WHO ARE IN CHARGE OF THE HUMANITARIAN RELIEF SECTOR. THEY'LL BE DOWN THERE FOR A LIMITED PERIOD OF TIME AND FOR A SPECIFIC MISSION, WHICH REALLY HAS THREE PARTS. ONE, TO STABILIZE THE SITUATION; SECOND, TO PREVENT A REOCURRENCE OF THE PROBLEM THAT WE HAD THE LAST COUPLE OF DAYS; AND THIRD, TO INVESTIGATE THE EVENTS THAT HAPPENED.

Q: THERE'S NO INDICATION IN THAT STATEMENT THAT WE'LL ATTEMPT TO REVERSE THE SITUATION WITH THE OCCUPATION OF PART OF KISMAYO BY

GENERAL MORGAN'S TROOPS.
A:— I'M NOT SURE THAT IT'S CORRECT TO SAY THERE WAS AN OCCUPATION OF KISMAYO BY GENERAL MORGAN'S TROOPS. I THINK THAT BADLY MIS STATES THE SITUATION BASED ON THE REPORTING WE'VE SEEN OUT OF MOGADISHU.
Q: CAN YOU CLARIFY THAT FOR US?
A: I THINK FRED PECK TALKED ABOUT WHAT HAPPENED, THIS CONFRONTA, TION THAT TOOK PLACE. BUT I THINK TO SAY THEY HAVE OCCUPIED KISMAYO IS NOT CORRECT. IN THE FIRST PLACE, YOU'RE TALKING ABOUT

/****** BEGINNING OF SECTION 004 *******/
GROUPS OF A COUPLE OF HUNDRED PEOPLE. YOU CAN'T OCCUPY A CITY OF 160,000 WITH A COUPLE OF HUNDRED PEOPLE.O IT JUST DOESN'T WORK THAT WAY.
THAT DOESN'T MEAN THERE ISN'T A PROBLEM. THAT DOESN'T MEAN THERE HAVEN'T BEEN POTENTIAL CLASHES AND INCIDENTS WHERE IN THIS CASE, BASICALLY TWO SIDES APPROACHED EACH OTHER WITH THE POSSIBILITY OF AN INCIDENT. I DON'T MEAN TO SAY THAT, BUT TO USE THE PHRASE "OCCUPY THE CITY" IS A GROSS OVERSTATEMENT OF WHAT HAPPENED.
Q: THE IG THE OTHER DAY SAID THAT THE C-17 PROGRAM SHOULD BE HELD UP UNTIL DOD DOES A COMPLETE REVIEW. IS THERE ANY PENTAGON REAC- TION TO THAT, OR ANY PLANS THAT ARE IN PLACE TO CONDUCT A REVIEW AND WHAT SORT OF REVIEW THAT WOULD BE?
A: ON THE C-17?
Q: YES.
A: THERE IS AN ONGOING PROCESS AS A RESULT OF THE IG REPORT. THE ACTING SECRETARY OF THE AIR FORCE, MICHAEL DONLEY, HAS BEEN DIRECTED TO RESPOND TO THE FINDINGS OF THE IG. AS YOU KNOW, THE IG REPORT, TALKED ABOUT SOME AIR FORCE OFFICIALS ACTING IMPROPERLY WITH HANDLING THE C-17 CONTRACT. IN ADDITION, THE FY93 AUTHORIZA, TION ACT REQUIRED THE SECRETARY OF DEFENSE, ACTING THROUGH THE UNDER SECRETARY FOR ACQUISITION, ESTABLISH AN INITIATIVE FOR THE C-17 TO MAINTAIN CONTROL OF OUR COSTS, CONTRACTOR PERFORMANCE, AND MANAGEMENT PERFORMANCE. THEY'RE WORKING ON THAT INITIATIVE NOW.
Q: SO IS DOD CONFIDENT THE WINGS CAN MEET THE SPECIFICATIONS?
A: I THINK WE WANT TO MOVE FORWARD ON THE PROGRAM AND TRY TO RESOLVE THE PROBLEMS. THE AIR FORCE BELIEVES THAT THESE ARE SOLVABLE PROBLEMS AND THAT IT WILL BE A SUCCESSFUL AIRPLANE' THE BUDGET TAPES WERE SENT TO OMB ON MARCH 9TH. SECOND, ON THE QUESTION OF THE CONVOYS, OVERLAND CONVOYS BEING ALLOWED TO PROCEED TO VARIOUS PLACES IN EASTERN BOSNIA, THE UN IS THE OFFICIAL SOUECE ON IT. THE UN TELLS US THAT THEY HAVE HAD CONVOYS GOING TO TUZLA AND ZEPA, BUT WE DON'T HAVE ANY RESULTS FROM THE ONE TO SREBRENICA YET. HE ASKED ME TO REFER YOU ALL TO THE UN.
Q: FOR THE RECORD, WHEN SECRETARY CHENEY WAS HERE AS DEFENSE SECRETARY, THERE WAS A THING CALLED PRINCIPLES OF INFORMATION. IT'S BEEN POSTED IN THE BUILDING. IT BEARS HIS SIGNATURE. WHERE DO THESE STAND WITH THE NEW SECRETARY OF DEFENSE?
A: IN WHAT SENSE? ARE WE GOING TO REISSUE THEM OVER HIS SIGNA- TURE?
Q: ARE THEY GOING TO BE RE-SIGNED, WILL THEY BE CHANGED, WILL THEY

REMAIN IN FORCE?
A:—THEY WILL REMAIN IN FORCE. ALL THESE DIRECTIVES REMAIN IN FORCE UNTIL THEY'RE CHANGED. YOU HAVE TO TAKE AFFIRMATIVE ACTION TO CHANGE DIRECTIVES OR THINGS OF THIS NATURE. I BELIEVE HE'S COMMITTED TO THEM, BUT WHETHER HE'LL REISSUE THEM OVER HIS OWN NAME, I DON'T KNOW. I'LL HAVE TO LOOK INTO THAT.
PRESS: THANK YOU.
ADMIN
BT

#1281

NNNN

INQUIRE=DOC16D
ITEM NO=00201140
ENVELOPE
CDSN = LGX009 MCN = 91317/10944 TOR = 913170807
RTTUZYUW RUEKJCS3402 3170806-UUUU--RUEALGX.
ZNR UUUUU
HEADER
R 130806Z NOV 91
FM JOINT STAFF WASHINGTON DC
INFO RUEALGX/SAFE
R 130727Z NOV 91
FM SECDEF WASHINGTON DC//ASD:PA//
TO AIG 8798
AIG 8799
RXFBI/AFCENT BRUNSSUM NL
ACCT DA-BHCWAA
BT
CONTROLS
UNCLAS , NATO UNCLASSIFIED FOR NATO ADDRESSEES
SECTION 01 OF 07

/********** THIS IS A COMBINED MESSAGE **********/
BODY
SUBJ: DOD NEWS BRIEFING
DELIVER DURING NORMAL DUTY HOURS
FOLLOWING IS TRANSCRIPT OF NEWS BRIEFING CONDUCTED BY MR. BOB HALL, DASD/PUBLIC AFFAIRS, ON TUES., NOV., 12, 1991 AT AT NOON:
I'D LIKE TO WELCOME FOUR JOURNALISM STUDENTS FROM THE AMERICAN UNIVERSITY -- FROM OUR REGULAR PROGRAM ,- AS WELL AS PROFESSOR SPEAR WHO IS SPONSORING 28 STUDENTS ATTENDING THE WASHINGTON SEMESTER PROGRAM. WE ALSO, I UNDERSTAND, HAVE A DOZEN STUDENTS FROM GEORGE WASHINGTON UNIVERSITY -- ALL HERE TO SEE ONE ELEMENT OF THE NEWS BUSINESS AND HOW WE DO PUBLIC INFORMATION.
YOU HAVE A BLUE TOP TODAY THAT WILL ANNOUNCE THAT WE'RE ENDING OR REDUCING OPERATIONS AT 71 ADDITIONAL SITES IN EUROPE BY 1995. THIS BRINGS THE TOTAL OF U.S. SITES IN EUROPE THAT HAVE ENDED OR REDUCED OPERATIONS, OR HAVE BEEN PLACED IN STANDBY STATUS, TO A TOTAL OF 381 FACILITIES. THAT'S A MORE THAN 25 PERCENT REDUCTION IN U.S. INSTALLATIONS THAT ARE BEING USED IN EUROPE. THERE ARE AN ADDITIONAL 24 BASES AT OTHER LOCATIONS OUTSIDE OF EUROPE, MAKING A GRAND TOTAL OF 405 WHERE WE HAVE REDUCED OR ENDED OPERATIONS. THAT'S A TOTAL OF 24 PERCENT, FOR THOSE OF YOU THAT ARE INTERESTED IN STATISTICS.
I SHOULD ALSO NOTE FOR YOU, IN THE BLUE TOP IN THE FOURTH PARA, GRAPH, THERE ARE 17 SITES CITED THAT WE HAVE NOT MENTIONED BEFORE BUT WE HAVE, IN FACT, TAKEN SOME ACTION ON. ANY QUESTIONS ON THE BASES?
I'D LIKE TO ANNOUNCE TODAY THAT WE WILL BE CONDUCTING THIS WEEK, IN COOPERATION WITH THE DEPARTMENT OF STATE AND THE AGENCY FOR NATIONAL DEVELOPMENT, THE 100TH AFGHAN RELIEF MISSION TO PAKISTAN. THIS WILL BE DURING THE WEEK OF NOVEMBER 13TH TO 19TH. THE FLIGHT

WILL DELIVER APPROXIMATELY 20,000 POUNDS OF DOD EXCESS COLD WEATHER GEAR -- CLOTHING, SLEEPING BAGS, AND BLANKETS -- AND WILL EVACUATE 27 WOUNDED AFGHANS AND THEIR ESCORTS TO EUROPE AND THE UNITED STATES FOR ADVANCED MEDICAL TREATMENT. THERE WILL BE AN MFC FOR YOU LATER TODAY.

WE HAVE ANOTHER MFC ON TWO REMAINS THAT HAVE BEEN RECOVERED DURING JDINT EXCAVATION EFFORTS BETWEEN THE U.S. AND THE LAOTIAN GOVERNMENTS. THESE WERE SERVICEMEN WHO WERE LOST IN A FEBRUARY 18, 1971 HELICOPTER CRASH. WE HAVE SPECIALIST 4TH CLASS ROBERT J. ENGEN AND SERGEANT WALTER E. LEWELLEN. THE REMAINS OF THESE AMERICANS WILL DEPART HICKAM AIRFORCE BASE IN HAWAII WITH A FULL MILITARY HONORS CEREMONY AT 8:15 HAWAII TIME, NOVEMBER 13TH. THEY'LL TRAVEL TO TRAVIS AIR FORCE BASE, CALIFORNIA, FOR THE FINAL JOURNEY HOME.

SECRETARY OF DEFENSE DICK CHENEY WILL SPEAK TO THE TOWN HALL OF CALIFORNIA AT 9:30 A.M. PACIFIC TIME WEDNESDAY, NOVEMBER 13TH, IN THE CRYSTAL ROOM OF THE BILTMORE HOTEL, 506 SOUTH GRAND AVENUE, LOS ANGELES. THE SPEECH WILL BE FOLLOWED BY QUESTIONS AND ANSWERS AND IT'S OPEN TO THE MEDIA. HE WILL ALSO HAVE A MEDIA AVAILABILITY PRIOR TO THE SPEECH AT 9:10 A.M. IN THE TIFFANY ROOM AT THE BILTMORE HOTEL. THE POINT OF CONTACT IS KAREN BURGESS, THE DIRECTOR OF COMMUNICATIONS FOR TOWN HALL AT (213)628-8141.

THE SAME EVENING SECRETARY CHENEY WILL ACCEPT THE ALBERT SCHWEITZER LEADERSHIP AWARD AND MAKE REMARKS AT THE HUGH O'BRIEN YOUTH FOUNDATION DINNER AT 7:30 P.M. IN THE INTERNATIONAL BALLROOM OF THE BEVERLY HILTON HOTEL, 9876 WILSHIRE BOULEVARD, LOS ANGELES. AGAIN, THE EVENT IS OPEN TO THE MEDIA. IT WILL BE PRECEDED BY A PHOTO OP AT 6:00 P.M' IN THE VERSAILLES ROOM OF THE HOTEL. THE POINT OF CONTACT IS MICKEY CROCKER AT (213)474,4370,EXTENSION 326.

I'D LIKE TO MAKE A FEW COMMENTS ON THE REPORTS ABOUT THE BLOOD DONATIONS. I THINK YOU PROBABLY HAVE THE MFC THAT WE HAVE ON THIS. ON FRIDAY, NOVEMBER 8TH, DR. ENRIQUE MENDEZ, THE ASSISTANT SECRETARY FOR HEALTH AFFAIRS, MADE A DECISION TO RECOMMEND THAT ALL DOD PERSONNEL WHO WERE DEPLOYED TO THE GULF AREA -- INCLUDING THE COUNTRIES OF SAUDI ARABIA, KUWAIT, IRAQ, BAHRAIN, QUATAR, THE UNITED ARAB EMIRATES, OMAN, AND YEMEN -- SINCE AUGUST 1, 1990, THAT THEY TEMPORARILY REFRAIN FROM DONATING BLOOD. THIS DECISION IS BASED ON THE DISCOVERY, SINCE THE GULF CONFLICT, OF A LIMITED NUMBER OF CASES OF A DISTINCT MEDICAL SYNDROME INVOLVING AN INFECTION WITH A PARASITE TRANSMITTED BY THE BITE OF SAND FLIES. THAT PARASITE IS KNOWN AS LEISHMANIA.

TO DATE, ONLY 22 CASES HAVE BEEN DISCOVERED AMONG THE MORE THAN HALF MILLION TROOPS WHO DEPLOYED TO THE GULF REGION, AND NONE OF THESE 22 CASES INVOLVE LIFE THREATENING ILLNESSES. THE ORGANISM USUALLY CAUSES AN EASILY TREATED SKIN DISEASE, AND THAT IS THE SITUATION IN 15 OF THE CASES. BUT DOCTORS AT WALTER REED ARMY MEDICAL CENTER AND THE WALTER REED ARMY INSTITUTE OF RESEARCH HAVE IDENTIFIED THE INFECTION VIA BONE MARROW CULTURE IN SEVEN PATIENTS WHO HAVE NO SKIN LESIONS. THESE SEVEN PATIENTS HAVE A MILD ILLNESS INVOLVING FEVER AND DIARRHEA.

MILITARY MEDICAL PERSONNEL ARE NOW TRYING TO DETERMINE HOW PREVALENT THE DISEASE MAY BE AMONG RETURNING SERVICE MEMBERS.

ALTHOUGH THE DOCTORS BELIEVE THE NUMBER OF CASES ARE SMALL, THEY WANT TO ENSURE THAT ALL THE CASES ARE QUICKLY DETECTED AND TREATED. WHILE THAT PROCESS IS UNDERWAY, DR. MENDEZ HAS MADE THE PARALLEL DECISION REGARDING BLOOD DONATIONS. THESE FORMS OF LEISHMANIASIS ARE NOT CONTAGIOUS IN PERSON,TO-PERSON CONTACT.

ALTHOUGH THERE HAVE BEEN NO CASES OF THIS SPECIFIC FORM BEING TRANSMITTED THROUGH BLOOD TRANSMISSIONS, FIVE CASES OF A RELATED SPECIES OF PARASITE HAVE BEEN REPORTED IN MEDICAL LITERATURE AS BEING TRANSMITTED THROUGH TRANSFUSION OF CONTAMINATED BLOOD. THEREFORE, THE DELAY IN MAKING BLOOD DONATIONS WILL ENABLE MEDICAL RESEARCHERS TO DETERMINE THE LEVEL OF ADDITIONAL INFECTIONS IN THE EXPOSED POPULATION, AND WILL ALSO ALLOW THEM TO DEVELOP A SCREENING TEST FOR INFECTION.

AS THE MFC SAYS, DOD OFFICIALS ARE WORKING CLOSELY WITH THE FOOD AND DRUG ADMINISTRATION, THE CENTER FOR DISEASE CONTROL, THE AMERICAN RED CROSS, THE AMERICAN ASSOCIATION OF BLOOD BANKS, THE COUNCIL OF CDMMUNITY BLOOD CENTERS, AND O7HER GROUPS INVOLVED IN THE COUNTRY'S BLOOD SUPPLY TO DECIDE HOW BEST TO HANDLE THE SITUATION.

Q: YOU MENTIONED THERE'S A SCREENING TEST BEING DEVELOPED. DOES THAT MEAN THAT PEOPLE WHO HAVE BEEN TO THE PERSIAN GULF AREA AND RETURNED WHO MAY HAVE SOME ANXIETY AS TO WHETHER THEY'RE CARRYING THE ILLNESS HAVE NO PRESENT WAY OF FINDING OUT THROUGH A SIMPLE TEST? OR IS THERE AN EXISTING TEST?

A: AT THIS TIME MY UNDERSTANDING IS WE DO NOT HAVE AN EXISTING TEST. THAT'S WHAT THEY'RE WORKING ON TRYING TO DEVELOP.

Q: WERE THERE ANY CASES THAT WERE MORE SERIOUS THAN THE FIVE WHERE THE PEOPLE EXPERIENCED DIARRHEA AND OTHER DISCOMFORTS?

A: SEVEN CASES. NO. AS I WENT THROUGH THIS WITH THE EXPERTS, THERE ARE SEVERAL CLINICAL FORMS, SEVERAL KINDS OF LEISHMANIASIS. ONE IS AN ULCER OR SKIN LESIONS WHICH ESSENTIALLY CAN BE TREATED VERY EASILY. BUT IF IT WASN'T TREATED, IT WOULD PRESUMABLY HEAL BY ITSELF. THAT'S CAUSED BY LIESMANIA TROPICA, WHICH IS WHAT WE'VE

/****** BEGINNING OF SECTION 002 ******/

IDENTIFIED IN THIS CASE. NORMALLY THAT HAS SKIN ULCERS. HOWEVER, IN THESE SEVEN CASES THEY'VE IDENTIFIED IT IN A DIFFERENT FORM. THERE IS ANOTHER KIND OF CLINICAL FORM, AND THAT IS LIESMANIA DONAVANI WHICH IS NORMALLY WHAT THEY CALLVISCERAL MEANING IN THE BLOOD MARROW OR THE SPLEEN OR OTHER ORGANS. THAT IS POTENTIALLY FATAL, BUT NOT IF IT'S TREATED, AND THERE ARE REGULAR TREATMENTS FOR IT. IN FACT, OUR PEOPLE HAVE DEALT WITH A NUMBER OF CASES OF IT IN THE PAST. BUT AS I SAY, THAT IS NOT THE KIND THEY'RE DEALING WITH NOW. THEY'VE CHEMICALLY ID'D IT AS LIESMANIA TROPICA, WHICH IS NORMALLY FOUND AS A SKIN...

Q: THE SEVEN INDIVIDUALS WHO HAD THE MORE SERIOUS MANIFESTATION OF THE LESS SERIOUS DISEASE...

A: A DIFFERENT MANIFESTATION.

Q: HOW MANY OF THEM ARE CURED? OR DO THEIR SYMPTOMS PERSIST AT THIS TIME?

A: I'LL HAVE TO CHECK AND SEE WHETHER ANYBODY IS CURED FROM THAT

GROUP.
Q: HOW LONG DOES IT TAKE FOR THE DISEASE TO MANIFEST ITSELF?
A: WHAT I MIGHT TRY TO DO, WE'VE GOT A FEW PEOPLE AVAILABLE TO ANSWER YOUR QUESTIONS. IF YOU'D LIKE, I CAN ASK THEM TO COME UP NOW AND MAYBE WE CAN TAKE SOME OF THESE.
Q: DO YOU KNOW WHERE THESE 22 WERE STATIONED ,- IN A SPECIFIC COUNTRY, IN A SPECIFIC AREA? WERE THEY STATIONED ALL OVER?
A: I DON'T KNOW. THEY WERE STATIONED INTHE REGION. MAYBE WE COULD ASK THE COLONEL IF HE'D LIKE TO COME ON UP. LET ME TAKE GENERAL QUESTIONS FIRST, AND THEN WE'LL GET INTO SOME SPECIFIC, TECHNICAL QUESTIONS.
Q: WITH SO MANY TROOPS HAVING BEEN PERSIAN GULF VETERANS, HOW MUCH BLOOD IS ON HAND IN RESERVE, AND WHEN WILL WE START TO CUT INTO THAT IN TERMS OF...
A: I'M TOLD THE EFFECT ON THE BLOOD SUPPLY IS MANAGEABLE. OBVIOUSLY, IT'S SOMETHING YOU'D RATHER NOT HAVE TO DO. THE MILITARY COMMUNITY IS ONE OF THE BIGGEST DONORS OF BLOOD. WE'D RATHER NOT CUT 500,000 OF THEM OUT OF THE DONATION PROCESS. BUT IT IS A MANAGEABLE PROBLEM.
Q: I'LL REPEAT THE QUESTION. IT WAS HOW LONG WOULD IT TAKE FOR THE DISEASE TO MANIFEST ITSELF. IN OTHER WORDS, HOW LONG SHOULD PEOPLE WHO WERE IN THE PERSIAN GULF CONTINUE TO WORRY THAT THEY MAY COME DOWN WITH THIS DISEASE?
COL OSTER: I'M COLONEL CHARLES OSTER. I'M THE CHIEF OF INFECTIOUS DISEASE AT THE WALTER REED ARMY MEDICAL CENTER, AND I'M THE CONSULTANT TO THE ARMY SURGEON GENERAL FOR INFECTIOUS DISEASES.
THE ANSWER TO THAT QUESTION IS THAT THE USUAL INCUBATION PERIOD FOR LEISHMANIASIS IS IN THE ORDER OF MONTHS, BUT PROLONGED INCUBATION PERIODS OF ONE TO TWO YEARS HAVE BEEN DESCRIBED FOR VISCERAL LEISHMANIASIS. SO I THINK WE WOULD CONTINUE TO HAVE SOME CONCERN FOR THE NEXT TWO YEARS IN OUR RETURNING SOLDIERS.
Q: WOULD A PERSON KNOW THAT HE OR SHE'S BEEN BITTEN BY A SAND FLY AND THEREFORE...
OSTER: SAND FLIES ARE ONLY A COUPLE OF MILLIMETERS LONG. THEY USUALLY BITE AT NIGHT. THEY'RE USUALLY NOT SEEN BY PATIENTS WHO ARE BITTEN.
Q: DO YOU KNOW WHETHER THESE 22 SOLDIERS, WHETHER THEY WERE IN THE SAME AREA, DIFFERENT AREAS OF THE PERSIAN GULF?
OSTER: MANY OF THESE SOLDIERS WERE IN UNITS THAT TRAVELED EXTENSIVELY THROUGHOUT NORTHEAST SAUDI ARABIA, KUWAIT, AND SOUTHERN IRAQ. OTHER UNITS WERE STATIONARY IN NORTHEAST SAUDI ARABIA -- ONE UNIT FROM DHAHRAN AND ONE UNIT FROM RIYADH. SO WE CANNOT, BASED ON OUR EPIDEMIOLOGY ON THESE FEW CASES, WE CANNOT PINPOINT A FULL ACCOUNTING FOR THE INFECTIONS.
Q: WHAT IS THE TREATMENT FOR THIS?
OSTER: THE TREATMENT IS A STANDARD TREATMENT THAT WAS DEVELOPED SOME 50 YEARS AGO WITH WHAT IS CALLED A PENTAVALENT ANIMONY COMPOUND. THE PRODUCT WE USE IS SODIUM STIBOGUCHNATE. THE TRADE NAME IS PENTOSTAM. IT'S MANUFACTURED BY THE WELCOME TRUST IN THE UK. WE GET OUR DRUG FROM THEM. IT'S A DRUG THAT HAS NOT BEEN LICENSED IN THIS COUNTRY BECAUSE THERE ARE ESSENTIALLY NO CASES OF &VIS,

CERAL) LEISHMANIASIS IN THE UNITED STATES, SO THERE'S BEEN NO DRUG COMPANY WILLING TO PAY THE PRICE TO GET A LICENSE. SO WE HAVE IT ON AN INVESTIGATIONAL NEW DRUG PROTOCOL FROM THE FDA' WE HAVE HAD EXTENSIVE EXPERIENCE TREATING LEISHMANIASIS IN OUR SOLDIERS OVER THE PAST 13 OR 14 YEARS'

Q: CAN YOU DESCRIBE WHETHER YOU TAKE THIS STUFF ORALLY, OR IS IT INTRAVENOUS?

OSTER: THE TREATMENT IS INTRAVENOUS, AND WE RECOMMEND FOR THE CUTANEOUS DISEASE 20 DAYS OF A SINGLE INTRAVENOUS INJECTION; AND FOR THE DISEASE THAT HAS GONE TO THE DEEP ORGANS, SO-CALLED VISCERAL DISEASE -- WE RECOMMEND A 30 DAY COURSE OF TREATMENT.

Q: YOU'RE HOOKED UP TO IT ONCE A DAY FOR 30 DAYS OR...

OSTER: WE CAN JUST PUT A NEEDLE IN THE VEIN, TAKE ABOUT 20 MINUTES TO RUN THE DRUG IN, PULL THE NEEDLE OUT, AND THE PATIENT CAN GO ON HIS WAY.

Q: CAN YOU TELL US A LITTLE BIT MORE ABOUT HOW SERIOUS THIS IS, IF IT IS STABLE, AND WHAT THE TYPE OF DISEASE IS, AND ALSO, BESIDES THE MEDICATION, ANYTHING ELSE THAT...

OSTER I THINK THE REASON WE'VE COME OUT WITH IT AS WE HAVE IS TO PROSCRIBE BLOOD DONATIONS IN THOSE SOLDIERS THAT HAVE BEEN IN THE MIDDLE EAST, BECAUSE THAT IS ONE THING WE WOULD LIKE TO PREVENT IS ANY CHANCE FOR CONTAMINATION OF THE BLOOD SUPPLY.

THE DISEASE THAT WE ARE DESCRIBING IS AN UNUSUAL DISEASE IN THAT IT'S CAUSED BY AN ORGANISM, EL TROPICA, WHICH NORMALLY CAUSES THE CUTANEOUS FORM OF THE DISEASE. HOWEVER, IN SEVEN OF THESE PATIENTS WE'VE GOTTEN IT FROM THE BONE MARROW WHICH IS INDICATIVE OF VISCERAL LEISHMANIASIS. BUT THE CLASSIC VISCERAL LEISHMANIASIS IS CAUSED BY A DIFFERENT ORGANISM CALLED L DONOVANI. NOW L. DONOVANI CAN CAUSE A FATAL ILLNESS IF UNTREATED. IT CAUSES ENLARGED LIVER, ENLARGED SPLEEN, CAUSES DEPRESSION OF THE RED CELL, WHITE CELL, AND PLATELET COUNTS, IT CAUSES IMMUNE SUPPRESSION. THOSE PEOPLE WHO GET ADVANCED FORMS OF THIS DISEASE WASTE AWAY WITH WEIGHT LOSS, LOW GRADE FEVER, AND EVENTUALLY PROBABLY DIE OF ANOTHER CAUSE, USUALLY A SECONDARY INFECTION -- PNEUMONIA, MEASLES, OR OCCASIONALLY THEY'LL DIE OF BLEEDING COMPLICATIONS. BUT IF THE DISEASE IS RECOGNIZED, IT IS VERY TREATABLE. THE MORTALITY RATE CAN GO FROM 90 PERCENT UNTREATED TO ONE OR TWO PERCENT WHEN TREATMENT IS USED.

Q: YOU MENTIONED A TWO YEARS PERIOD OF PRIMARY CONCERN. IS THERE SOME RECURRENCE TO THIS AFTER AWHILE IN SOME CASES?

OSTER: I DON'T KNOW THE ANSWER TO THAT, PRECISELY. WE CAN MAKE AN ANALOGY TO THE VISCERAL LEISHMANIASIS CAUSED BY L. DONOVANI. L. DONOVANI NOW HAS BEEN RECOGNIZED IN EUROPE, IN SOUTHERN EUROPE -- FRANCE, SPAIN, AND ITALY -- AS RELAPSING WHEN PATIENTS DEVELOP AIDS. IT'S ALSO BEEN RECOGNIZED IN PATIENTS WHO HAVE HAD RENAL TRANSPLANTS OR OTHER TRANSPLANTS WHO ARE ON IMMUNOSUPPRESSION.

/****** BEGINNING OF SECTION 003 ******/

IT'S BEEN RECOGNIZED IN PATIENTS WHO ARE GETTING CANCER CHEMOTHERAPY. SO THAT THERE IS A POTENTIAL FOR RELAPSE WHEN A PERSON DEVELOPS AN IMMUNE DEFICIENCY.

HALL: JUST TO CLARIFY AGAIN, L. DONOVANI IS A DIFFERENT...

OSTER: AS I SAID, THIS IS AN ANALOGY TO L. DONOVANI, A DIFFERENT ORGANISM. WE DON'T KNOW HOW EL TROPICA IS GOING TO BEHAVE.
Q: WHAT ARE THE SYMPTOMS, AND HOW DID YOU FIND THE INITIAL CASES?
OSTER: WHEN WE WENT INTO OPERATION DESERT SHIELD IN AUGUST OF 1990, WE SAT DOWN AND REVIEWED EXTENSIVELY THE LITERATURE OF THE ENDEMIC DISEASES IN THAT AREA OF THE WORLD. WE PUBLISHED A HAND BOOK WHICH WAS DISTRIBUTED TO ALL OF OUR SOLDIERS, ALL OF OUR MEDICAL PERSONNEL IN SUPPORT OF DESERT SHIELD. AS A CONSEQUENCE, OUR MEDICAL PEOPLE WERE ALERTED TO DISEASES THAT THEY WOULDN'T OTHERWISE HAVE MUCH KNOWLEDGE OF.
WE ALSO PUBLISHED AN ARTICLE IN THE NEW ENGLAND JOURNAL OF MEDICINE IN MARCH REVIEWING THE SAME SUBJECT. AND NAVY PHYSICIANS PUBLISHED AN ARTICLE IN THE REVIEWS OF INFECTIOUS DISEASES IN JANUARY OF THIS YEAR. SO WE HAD A HEIGHTENED AWARENESS OF PROBLEMS THAT WERE POTENTIAL TO OCCUR IN OUR SOLDIERS IN THE GULF.
BECAUSE OF THIS, A SOLDIER ABOUT AYEAR AGO DEVELOPED A HIGH FEVER WHICH WAS UNEXPLAINED. A VERY ASTUTE PHYSICIAN THOUGHT ABOUT THE POSSIBILITY OF LEISHMANIASIS AND ORDERED AN ANTIBODY TITER WHICH CAME BACK POSITIVE. THEN THAT PHYSICIAN MEDIVAC'D HIS PATIENT BACK TO US AT WALTER REED FOR FURTHER CONFIRMATION OF THE DIAGNOSIS AND TREATMENT. WE DIDN'T SEE ANOTHER CASE UNTIL APRIL OF THIS YEAR, AGAIN, PRESENTED WITH HIGH FEVERS, PRIMARILY, WHICH WAS UNEXPLAINED BY ANY OTHER ILLNESSES. WE DID A BONE MARROW ON HIM AND WERE ABLE TO ESTABLISH A DIAGNOSIS OF LEISHMANIASIS.
THE OTHER CASES, TWO MORE PRESENTED WITH HIGH FEVERS THAT WERE UNEXPLAINED, AND TWO OTHERS PRESENTED WITH CHRONIC ILLNESS, WITH LOW GRADE FEVERS, ABDOMINAL COMPLAINTS, PRIMARILY WATERY DIARRHEA AND ABDOMINAL CRAMPY PAIN WHICH EVOLVED INTO ENLARGEMENT OF THE ORGAN , THE SPLEEN AND THE LIVER, MORE LIKE THE CLASSICAL VISCERAL LEISHMANIASIS.
SO WE HAVE A SPECTRUM OF DISEASE, FROM AN ACUTE TYPE OF ILLNESS TO A MORE CHRONIC DISEASE WITH ABDOMINAL COMPLAINTS. ONE OF OUR PATIENTS WAS, IN FACT, ASYMPTOMATIC. WE IDENTIFIED HIM BY GOING TO THE UNIT OF AN INDEX CASE AND TALKING TO THOSE SOLDIERS AND DOING THE ANTIBODY TESTS FOR THE LEISHMANIA ANTIBODY, AND THEN IN SOME OF THOSE SOLDIERS WHO HAD HIGH ANTIBODY TITERS, WE ASKED THEIR PERMISSION TO DO A BONE MARROW, AND ONE OF THOSE PATIENTS WAS POSITIVE ON BONE MARROW. HE IS ASYMPTOMATIC. HE REMAINS ASYMPTOMATIC. ALL WE'VE ELECTED TO DO WITH HIM IS TO SEE HIM ON A REGULAR BASIS, TO BE SURE THAT HE DOESN'T BECOME ILL. WE HAVE NOT TREATED HIM WITH PENTOSTAM.
Q: REGARDING YOUR CONCERN FOR THE BLOOD SUPPLY, IT'S BEEN SEVERAL MONTHS SINCE THE FIRST TROOPS CAME BACK FROM THE GULF. ISN'T IT POSSIBLE THAT SOME OF THEM HAVE ALREADY DONATED BLOOD? DO YOU HAVE ANY (INFORMATION) ON THAT?
OSTER: SIR, I'M NOT THE RIGHT PERSON TO ANSWER THAT QUESTION. I'M NOT A BLOOD BANKER. GENERAL BLANCK, DO YOU WANT TO TAKE THAT?
Q: OF THE 22 CASES THAT YOU'VE HAD, DO ANY OF THOSE CONTINUE NOW UNDER TREATMENT AT WALTER REED, OR HAVE THEY ALL BEEN SUCCESSFULLY TREATED AND RELEASED? WHAT'S THEIR STATUS?
OSTER: OF THE SEVEN PATIENTS WE'VE IDENTIFIED, FIVE HAVE BEEN

SYMPTOMATIC AND BEEN TREATED, AND ALL HAVE RESPONDED TO THERAPY. ONE IS ASYMPTOMATIC. WE CONTINUE TO FOLLOW HIM. THE SEVENTH PATIENT IS A PATIENT WHO'S STILL AT WALTER REED, WHO HAS ANOTHER MEDICAL PROBLEM THAT'S HAD TO BE TAKEN CARE OF FIRST. SO WE DO PLAN TO TREAT HIM IF HE'S SYMPTOMATIC.

Q: ARE ALL THESE TREATMENTS DONE AT WALTER REED? WHEN YOU HAVE IT, DO YOU MAINTAIN YOUR ORDINARY, DAILY ROUTINE, OR ARE YOU HOSPITALIZED, CAN YOU CONTINUE TO WORK? WHAT HAPPENS?

OSTER: SEVERAL OF OUR PATIENTS HAVE HAD A CHRONIC FATIGUE BASICALLY. THEY HAVEN'T BEEN ABLE TO PERFORM UP TO THEIR NORMAL STANDARDS, SO THAT HAS BEEN A PROBLEM FOR A COUPLE OF OUR PATIENTS. THAT HAS GOTTEN BETTER WITH TREATMENT.

Q: IS EVERYBODY BROUGHT HERE TO WALTER REED, OR IF YOU'RE AT FORT BRAGG DO YOU GET TREATED AT FORT BRAGG?

OSTER: THE ARMY SURGEON GENERAL, BACK IN 1977, MADE A DECISION TO LOCALIZE ALL OF THE TREATMENT AND CARE FOR LEISHMANIASIS AT WALTER REED ARMY MEDICAL CENTER. AT THAT TIME, WE RECOGNIZED THE PROBLEM OF CUTANEOUS LEISHMANIASIS FROM TROOPS WHO WERE STATIONED IN PANAMA, SO WE WERE SEEING ULCERS IN OUR SOLDIERS WHO WERE GOING TO PANAMA. SINCE NOBODY HAD A STRONG CLINICAL EXPERIENCE WITH LEISHMANIASIS IN THIS COUNTRY, IT WAS ELECTED TO BRING IT TO ONE CENTER, SO AT LEAST A FEW DOCTORS COULD LEARN MORE ABOUT THIS DISEASE AND HOW TO TREAT IT AND APPROPRIATE MANAGEMENT. IT WAS BECAUSE OF THAT DECISION 13 YEARS AGO THAT WE WERE IN A POSITION TO SUPPORT ODS IN MAKING A DIAGNOSIS OF LEISHMANIASIS, BECAUSE WE HAD THE DOCTORS THAT KNEW HOW TO RECOGNIZE THE DISEASE AND RIGHT UP FROM THE BACK DOOR OF WALTER REED ARMY MEDICAL CENTER IS WALTER REED ARMY INSTITUTE OF RESEARCH WHERE THEY'RE DOING ALL THE RESEARCH ON LEISHMANIASIS.

Q: BUT IT DOES MEAN YOU HAVE TO COME UP TO WALTER REED FOR 20 OR 30 DAYS?

OSTER: THAT'S CORRECT.

Q: BASED ON WHAT YOU KNOW ABOUT THE INCLINATION OF THE DISEASE, IS THE APPEARANCE SO FAR OF SEVEN CASES OUT OF 500,000, IS THAT AN ALARMING INCIDENCE OF THE DISEASE?

OSTER: IT'S NOT ALARMING, CERTAINLY -- SEVEN OUT OF 500,000 CAN'T BE BLOWN UP INTO TOO BIG OF A PROBLEM. BWT WE DON'T KNOW WHAT THE ACTUAL NUMERATOR IS OF THE PATIENTS WHO HAVE BEEN INFECTED. WHAT WE'VE SEEN ARE PATIENTS WHO HAVE A NON-LIFE THREATENING (FEBRILE) ILLNESS WITH SOME CHRONIC COMPLAINTS THAT HAVE EVENTUALLY GOTTEN TO WALTER REED ARMY MEDICAL CENTER WHERE WE THOUGHT ABOUT LEISHMANIASIS AND WERE ABLE TO DIAGNOSE IT. THERE MAY HAVE BEEN MANY OTHER SOLDIERS WHO HAD A SIMILAR MILD ILLNESS THAT WERE NEVER DIAGNOSED AS HAVING LEISHMANIASIS. BUT I THINK THE IMPORTANT POINT IS THAT THIS SYNDROME APPEARS TO BE SELF LIMITED, EVEN WITHOUT TREATMENT. BY THAT I MEAN THE SOLDIERS THAT WE TREATED WERE ALREADY IMPROVING BEFORE TREATMENT. SO I THINK THERE IS LITTLE CONCERN HERE THAT WE'RE GOING TO HAVE SOLDIERS UNRECOGNIZED WHO WILL GO ON AND DEVELOP A MORE SERIOUS ILLNESS. I THINK WE WILL PROBABLY HEAR ABOUT MORE CASES THAT DID HAVE A FEVER, MAYBE DID HAVE SOME ABDOMINAL COMPLAINTS, MAYBE DID HAVE SOME EASY FATIGUABILITY FOR SOME

PERIOD OF TIME THAT THEN GOT BETTER. WE WILL ONLY DIAGNOSE THOSE PATIENTS IN RETROSPECT.
Q: YOU TALKED ABOUT CASES OF SOLDIERS WHO SERVED IN PANAMA IN THE LATE '70S. GIVEN THE LARGE NUMBER OF U.S. CIVILIAN AND MILITARY PERSONNEL WHO HAVE BEEN IN AND OUT OF THE PERSIAN GULF, INCLUDING DIPLOMATS, FOR MANY MANY YEARS, IS THIS THE FIRST TIME THAT THE

/****** BEGINNING OF SECTION 004 ******/
MILITARY HAD DETECTED LEISHMANIASIS FROM THE AREA?
OSTER: LEISHMANIASIS IS WELL KNOWN TO OCCUR IN THAT AREA OF THE WORLD. CUTANEOUS LEISHMANIASIS IS DESCRIBED IN ALL OF THE AREAS OF THE GULF. EL TROPICA CAUSES A LARGE NUMBER OF THOSE CASES. VISCERAL LEISHMANIASIS, THE CLASSICAL LESHMANIASIS CAUSED BY EL DANAVANI, WAS ONLY RECOGNIZED IN IRAQ, IRAN, YEMEN, AND IN THE EXTREME SOUTHWEST OF SAUDI ARABIA. SO THAT THE AREA OF OUR OPERATIONS, WE WERE REALLY NOT CONCERNED ABOUT THE POSSIBILITY OF VISCERAL LESHMANIASIS. SO THIS DID COME AS A SURPRISE TO US.
Q: ARE YOU WORKING WITH SAUDI OR KUWAITI AUTHORITIES (INAUDIBLE)?
OSTER: NO MA'AM. THERE ARE TWO ASPECTS OF THIS. ONE IS TO MAKE A DECISION ABOUT OUR PROBLEM RIGHT NOW AND OUR RETURNING TROOPS. THAT, AGAIN, HAS TWO RAMIFICATIONS. ONE IS THE BLOOD TRANSFUSIONS WHICH WE'VE CURTAILED; THE SECOND IS TO PROVIDE CLINICAL SUPPORT FOR DIAGNOSIS OF PATIENTS WHO MAY HAVE BEEN INFECTED WITH THIS. THE OTHER MAJOR AREA OF INTEREST IS DEFINING THE SYNDROME IN A RESEARCH MODE. PART OF THAT WOULD BE TO GO TO THE ENDEMIC AREA AND TALK TO PHYSICIANS THERE, EXAMINE PATIENTS, DO TESTS ON THEIR PATIENTS. BUT THAT'S AN ENTIRELY DIFFERENT PROJECT. BUT WE'RE VERY INTERESTED IN DEFINING THIS NEW DISEASE, IF YOU WILL, MORE CAREFULLY.
Q: TO CLARIFY, BECAUSE THE ANSWERS DON'T SEEM TO ME TO BE TOTALLY CONSISTENT. A RETURNING SERVICEMAN GETS OFF THE PLANE, SAYS I JUST DON'T KNOW IF I HAVE THIS OR NOT, I WANT TO BE TESTED. DO YOU HAVE A SCREENING PROCEDURE OR TEST THAT HE OR SHE CAN...
OSTER: YES. I'M GLAD YOU BROUGHT THAT UP AGAIN, BECAUSE THERE HAS BEEN TWO ANSWERS TO THAT QUESTION. THE CORRECT ANSWER IS YES. WE HAVE A TEST THAT'S AVAILABLE NOW. WE'VE SET UP A MECHANISM TO HANDLE REQUESTS. FIRST OF ALL, WE'VE SENT OUT A LETTER TO ALL PHYSICIANS IN THE DEFENSE DEPARTMENT. SECONDLY, WE'VE SENT OUT ANDTHER LETTER THROUGH THE FDA TO CIVILIAN PHYSICIANS, DESCRIBING WHAT WE HAVE FOUND AND SUGGESTING IF THEY HAVE A POTENTIAL CASE THEY CAN CONTACT US FOR ADVICE. FURTHERMORE, WE WILL PROVIDE THEM DIAGNOSTIC SUPPORT BY DOING A SERUM ANTIBODY TEST IF THEY WILL SEND US THE SERUM FROM THEIR PATIENT.
FURTHER, WE WOULD PROPOSE THAT IF THAT TEST WERE POSITIVE THAT WE SHOULD EITHER GET THE PATIENT DIRECTLY, OR WE SHOULD GET A SAMPLE OF THE BONE MARROW THAT WE CAN THEN TEST WITH ANOTHER, MORE SPECIFIC TEST, FOR THE PRESENCE OF INFECTION. IF THAT WERE THEN POSITIVE, WE WOULD RECOMMEND TWO COURSES OF ACTION. IF THE PATIENT HAS RECOVERED FROM HIS ILLNESS AND IS CURRENTLY ASYMPTOMATIC, THAT HE OR SHE JUST BE FOLLOWED ON A REGULAR BASIS. THE OTHER COURSE OF ACTION WOULD BE IF THAT PATIENT IS STILL SYMPTOMATIC, THAT WE WOULD

OFFER THEM TREATMENT WITH PENTOSTAM.
Q: WHAT'S IT CALLED?
OSTER: IT'S CALLED PENTOSTAM. THAT'S THE TRADE NAME.
Q: IS IT YOUR RECOMMENDATION THAT EVERYBODY WHO SERVED IN THE PERSIAN GULF BE TESTED FOR THIS WITH THE ANTIBODY TEST?
OSTER: NO SIR, WE CANNOT SUPPDRT THAT WITH CURRENTLY AVAILABLE TESTS. WE ARE WORKING TO DEVELOP TESTS LIKE AN ELIZA WHICH COULD BE DONE ON MANY MORE SAMPLES. OUR CURRENT TECHNOLOGY IS LIMITED TO WHAT'S CALLED AN INDIRECT FLUORESCENT ANTIBODY WHICH HAS TO BE READ MANUALLY WITH A FLUORESCENT MICROSCOPE. SO IT'S VERY HIGHLY LABOR INTENSIVE. BUT IF WE CAN CONVERT TO AN ELIZA FORMAT, THEN WE WOULD HAVE THE POTENTIAL, OBVIOUSLY, FOR SCREENING LARGER NUMBERS OF SOLDIERS.
Q: SO AT THIS POINT YOU WILL TEST PEOPLE WHO HAVE SYMPTOMS?
OSTER: THAT'S CORRECT. OUR CONCERN NOW IS A CLINICAL CONCERN. WE WANT TO BE ABLE TO IDENTIFY AND TREAT THOSE PATIENTS WHO ARE ILL WITH THIS DISEASE.
Q: WHEN YOU SAY IT COULD TAKE UP TO TWO YEARS FOR THE INCUBATION PROCESS TO TAKE HOLD, DOES THAT MEAN THAT OVER THE NEXT TWO YEARS NO ONE WHO'S SERVED IN OPERATION DESERT STORM SHOULD GIVE BLOOD?
OSTER: I DON'T THINK WE HAVE ENOUGH INFORMATION TO ANSWER THAT QUESTION NOW. THIS IS SOMETHING WE'D NEED TO DO RESEARCH ON, TO DEFINE THE NATURAL HISTORY DF THIS DISEASE, TO DEFINE THE MAGNITUDE OF THE PROBLEM -- THAT IS THE NUMBER OF PATIENTS WHO ARE ACTUALLY INFECTED, TO DEVELOP BETTER DIAGNOSTIC TESTS. THEN WE CAN MAKE A DECISION ABOUT HOW LONG TO DEFER FROM BLOOD DONORS. IT'S ENTIRELY CONCEIVABLE THAT WE MAY BE ABLE TO DEVELOP A DIAGNOSTIC TEST, SAY AN ELIZA, WHERE WE COULD SCREEN PATIENTS FOR THIS INFECTION, AND THEN ALLOW THEM TO DONATE IF THEY SCREEN NEGATIVE.
Q: BUT THE IMPLICATIONS OF IT MEANS THAT OVER THE NEXT TWO YEARS, PRESUMABLY, PEOPLE WHO HAVE NO SYMPTOMS RIGHT NOW COULD DEVELOP THOSE SYMPTOMS?
OSTER: AGAIN, I CAN'T ANSWER THAT. THE QUESTION I ANSWERED ABOUT TWO YEARS IS L. DONOVANI, WHICH IS THE CLASSICAL FDRM OF VISCERAL LESHMANIASIS. SO THE NATURAL HISTORY OF EL TROPICA INFECTIONS, WE DON'T KNOW. THE NATURAL HISTORY OF SKIN DISEASE CAUSED BY EL TROPICA USUALLY HEALS UP IN MONTHS -- EVEN WITHOUT TREATMENT. BUT WE DON'T KNOW WHAT WILL HAPPEN TO THIS FORM THAT WE'VE IDENTIFIED WHERE IT INFECTS THE BONE MARROW AND OTHER DEEP ORGANS. SO ALL OF THIS DATA I THINKWILL BE COMING OUT AS WE LEARN MORE ABOUT THIS DISEASE'
Q: (INAUDIBLE) ABOUT BLOOD DONATIONS. THIS EXTENDS TO ANYONE WHO'S BEEN OVER THERE, NOT JUST MILITARY PERSONNEL?
HALL: WHY DON'T WE MOVE ON TO THE QUESTION OF BLOOD SUPPLY.
Q: THE PENTOSTAM IS IMPORTED FROM BRITAIN?
OSTER: YES.
Q: SO I GATHER THEY'VE HAD MORE EXPERIENCE WITH IT. DO THEY HAVE A SCREENING TEST DEVELOPED?
OSTER: FIRST OF ALL, I DOUBT THAT THEY HAVE ANY MORE EXPERIENCE THAN WE DO. ONE OF THE ADVANTAGES OF WORKING IN THE MILITARY IS THAT WE DO HAVE OVERSEAS LABORATORIES WHERE WE ARE ABLE TO INVESTI-

GATE DISEASES SUCH AS LESHMANIASIS. IT WAS MY PERSONAL PRIVILEGE TO HAVE SERVED IN KENYA FOR FOUR YEARS WHERE I STUDIED VISCERAL LESHMANIASIS, AND TOOK CARE OF PATIENTS WITH CLASSICAL LESHMANIASIS DUE TO L. DONOVANI IN KENYA. SO ACTUALLY OUR MILITARY HAS PROBABLY THE MOST EXTENSIVE EXPERIENCE WITH LESHMANIASIS OF ANY GROUP IN THE WORLD.
Q: ARE YDU HANDLING ALL THE CASES, OR ARE THE SEPARATE SERVICES... YOU'RE JUST TALKING ABOUT ARMY PEOPLE NOW?
OSTER: THE CASES WE'VE IDENTIFIED SO FAR HAVE ONLY BEEN IN ARMY PERSONNEL. WE'VE SHARED DUR INFORMATION WITH THE NAVY...
Q: THEY MAY HAVE OTHER CASES -- THE NAVY, THE MARINES CORPS, THE AIR FORCE...
OSTER: I'M NOT AWARE OF ANY. I'VE TALKED WITH THE OTHER SERVICES, AND THEY HAVEN'T IDENTIFIED ANY CASES YET.
Q: ALL 22 ARE IN THE ARMY?
OSTER: YES.
HALL: WHY DON'T WE MOVE ON AND TALK A LITTLE BIT ABOUT THE BLOOD SUPPLY? GENERAL BLANCK?

/****** BEGINNING OF SECTION 005 ******/
BLANCK: (BRIGADIER GENERAL) RONALD BLANCK. I'M THE DIRECTOR FOR PROFESSIONAL SERVICES IN THE OFFICE OFTHE ARMY SURGEON GENERAL.
Q: ABOUT THE CONTAMINATION, SOLDIERS WHO HAVE RETURNED FROM SAUDI ARABIA OR ONE OF THOSE COUNTRIES WHO HAVE ALREADY GIVEN BLOOD SINCE THEY GOT HOME, AND BEFORE THIS SUSPENSION (WENT INTO EFFECT). WHAT ARE THE CHANCES THAT THE BLOOD SUPPLY HAS ALREADY BEEN CONTAMINATED WITH THIS PARASITE?
BLANCK: WE THINK IT VERY, VERY LOW. FIRST OF ALL, AS HAS BEEN ALLUDED TO, WE'VE ONLY SEEN SEVEN CASES OF THIS NEW FORM OF WHAT APPEARS TO BE A MILD NON,FATAL ILLNESS OUT OF ALL OF THOSE THAT HAVE BEEN THERE. IT IS THEORETICALLY POSSIBLE, INDEED, 1'HAT THERE ARE DTHER CASES OUT THERE WHO COULD HAVE DONATED BLOOD AND WHO THEREFORE COULD HAVE TRANSMITTED THIS PARASITE, BUT IT'S VERY, VERY SLIGHT. WE THINK WE HAVE, IN GOING AROUND THE SYSTEM, IDENTIFIED ANY OF THE CASES WHO HAVE REMAINED SYMPTOMATIC THAT WOULD HAVE COME TO OUR ATTENTION. THOSE THAT ARE CLEAR, THAT IS WHO ARE NOT SYMPTOMATIC ANY MORE, PRESUMABLY WOULD NOT BE TRANSMITTING THE PARASITE ANY MORE. IT IS RARE.
FINALLY, IN MOST OF OUR CASES, THERE WERE ELEVATIONS OF THE LIVER TESTS, AND THAT'S ONE OF THE SCREENING TESTS FOR BLOOD TRANSFUSION THAT WOULD HAVE PRECLUDED THEM FROM DONATING, SO WE DON'T THINK THAT VERY MANY, IF ANY, HAVE TRANSMITTED THIS ORGANISM IN THE BLOOD SUPPLY, AND WE DON'T THINK IT'S CONTAMINATED.
Q: WHAT ABOUT THE SUPPLY OF BLOOD? IF YOU TAKE 500,000 PLUS PEOPLE BUT OF THE POTENTIAL DONOR POOL, WHAT DO YOU DO TO THE SUPPLY?
BLANCK: THE SUPPLY IS GOING TO BE DECREMENTED BY THE AMOUNT THAT THEY WOULD DONATE. IN OUR SYSTEM, IN DEPARTMENT OF DEFENSE, THESE ARE EXACTLY THE PEOPLE THAT ARE OUR LARGEST POOL OF DONORS, SO WE WILL HAVE TO RELY MUCH MORE ON GETTING THE NON,DESERT STORM SOLDIERS, SAILORS, AIRMEN, AND MARINES, AS WELL AS RELYING ON CIVILIAN

BLOOD SUPPLIES FOR THE FORESEEABLE FUTURE, UNTIL WE DEFINE THIS SYNDROME.
Q: IS THERE A WAY OF QUANTIFYING THE LOSS? I THINK I HEARD YOU SAY SOMETHING LIKE FIVE PERCENT OF ALL MILITARY PEOPLE ARE BLOOD DONORS, IS THAT RIGHT?
BLANCK: IT'S ACTUALLY PROBABLY HIGHER THAN THAT, AND I CAN'T GIVE YOU A GOOD FIGURE. BUT THE ACTIVE DUTY POPULATION IS WHERE WE GET OUR BLOOD, AND THIS IS A LARGE PERCENT OF THAT...
Q: CAN YOU SAY WHAT PERCENTAGE OF THE NATION'S BLOOD SUPPLY COMES FROM THE MILITARY?
BLANCK: I DON'T KNOW, NO.
Q: THIS RECOMMENDATION AGAINST GIVING BLOOD EXTENDS NOT ONLY TO MILITARY BUT TO ANYONE ELSE WHO WAS IN THE GULF DURING THAT AREA?
BLANCK: I THINK THAT'S A FAIR STATEMENT. THE WAY WE'VE CHOSEN TO CONTROL IT IS PRESENTED THIS DATA TO THE OTHER SERVICES. THE THREE SERVICES SITTING TOGETHER WITH DEPARTMENT OF DEFENSE CAME UP WITH A POLICY. THAT POLICY IS THAT DEPARTMENT OF DEFENSE BLOOD DONOR CENTERS WILL NOT ACCEPT BLOOD TRANSFUSIONS OR DONATIONS FROM ANY OF THOSE WHO HAVE SERVED IN THE PERSIAN GULF AREA, THE COUNTRIES MENTIONED, SINCE 1 AUGUST 1990. THIS WOULD INCLUDE ANYONE WHO IS NOT A SERVICE MEMBER, WOULD ALSO BE PRECLUDED FROM DONATING IN THE DOD FACILITIES.
WE HAVE DISCUSSED THIS INFORMATION WITH THE CIVILIAN DONOR CENTERS -- THE RED CROSS AND SO FORTH, AND THEY ARE COMING UP WITH THEIR OWN POLICIES, AND I'M NOT QUITE SURE WHAT THOSE POLICIES WOULD BE, BUT I WOULD IMAGINE THEY WOULD BE VERY SIMILAR TO WHAT I'VE JUST DESCRIBED. BUT THAT'S THEIR DECISION, NOT OURS.
Q: COULD YOU EXPLAIN WHY AUGUST 1, 1990, WAS PICKED AS A CUTOFF DATE?
BLANCK: SIMPLY BECAUSE THAT'S WHEN WE BEGAN SENDING OUR LARGE BODY OF TROOPS THERE. UP UNTIL THEN THERE WERE CERTAINLY PERSONNEL THERE, BUT IT NUMBERED IN THE 100'S, AND WE SAW NO PROBLEMS PRIOR TO THIS. NOW THEY MAY HAVE GONE UNRECOGNIZED, I DON'T KNOW. BUT WE HAD TO CHOOSE SOME DATE AND THAT SEEMED REASONABLE.
Q: WHY WASN'T THE PRECAUTION AGAINST DONATING BLOOD TAKEN EARLIER, EVEN APRIL, AFTER THE SECOND CASE WAS CONFIRMED?
BLANCK: THAT'S A GOOD QUESTION. SIMPLY BECAUSE IT WAS SUCH A RARE INSTANCE, AND WE DID NOT KNOW OR THINK -- DID NOT KNOW IS THE TERM -- AND DID NOT THINK IT REPRESENTED A LARGER PROBLEM THAT WE NOW THINK POTENTIALLY IT MAY, WITH THE SEVEN CASES. SO IT HAD TO DO WITH WHETHER IT'S A SINGLE RARE INSTANCE, OR ONE OR TWO VERSUS THE POTENTIAL OF BEING A LARGER PROBLEM. ONCE YOU HAVE SEVEN CASES, I THINK YOU HAVE TO SAY IT'S POTENTIALLY A LARGER PROBLEM.
Q: IT SOUNDS LIKE THIS WORD GOING OUT TODAY WILL ALARM A LOT OF PEOPLE WHO SERVED IN THE GULF WHOMAY WANT TO BE TESTED JUST AS A PRECAUTION. I UNDERSTAND YOU DON'T HAVE THE BACKUP RIGHT NOW TO DO THAT SORT OF THING, BUT WOULD YOU FORESEE A PRECAUTIONARY TEST THAT VETERANS CAN TAKE DEVELOPING PRETTY QUICKLY?
BLANCK: YES. I SEE A TEST DEVELOPING RELATIVELY RAPIDLY. THAT SHOULD BE POSSIBLE. BUT I HOPE YOU WILL HELP US ON THIS. WE DON'T WANT TO SPREAD PANIC, BECAUSE THIS IS NOT THE KIND OF DISORDER THAT

WOULD WARRANT THAT. ONE, IT'S RARE. TWO, EVERY PIECE OF EVIDENCE WE HAVE SUGGESTS THAT IT IS SELF LIMITED. IT DOES NOT CAUSE LIFE THREATENING OR FATAL DISEASE. IT SHOULD NOT GO ON TO EVEN A CHRONIC DEBILITATING DISEASE, WE DON'T BELIEVE. AND FINALLY, IT'S EASILY TREATABLE. I WOULD ASK THAT AT THIS POINT, THAT INFORMATION BE PROVIDED SO THAT WE DON'T HAVE EVERYBODY.WHO SERVED OVER THERE WORRYING ABOUT THIS AND TRYING TO GET THEMSELVES TESTED. I REALLY DON'T THINK THAT'S APPROPRIATE. CERTAINLY, WE WILL DO THE STUDIES AND MAKE THE INFORMATION AVAILABLE IF THAT KIND OF THING COMES ABOUT.
Q: THERE WAS AN EARLIER QUESTION, AND I'D LIKE TO REPEAT IT. YOU HAVE 22 CASES DETECTED OUT OF HALF A MILLION. IS THAT A LOT, IS THAT AVERAGE, IS THAT LITTLE? IS THERE A WAY OF PUTTING THAT INTO. .
BLANCK: I THINK THAT'S A SMALL AMOUNT COMPARED TO WHAT YOU MIGHT SEE IN A PDPULATION THAT LIVES THERE. THAT HAS TO DO WITH THE OVERALL GOOD HEALTH OF THE SOLDIERS, THE PROTECTIVE MEASURES TAKEN -- USE OF INSECT REPELLANT, THAT SORT OF THING, AND THE FACT THAT THEY WERE THERE FOR A RELATIVELY SHORT TIME COMPARED TO A POPULATION THAT LIVES THERE. HOW WOULD YOU COMPARE THIS WITH TRAVELERS? IT'S DIFFICULT TO COMPARE. TRAVELERS MIGHT ONLY BE THERE DAYS OR WEEKS. SO I WOULD SAY THIS ISA SMALL NUMBER AND SOMETHING THAT WE WOULD EXPECT TO SEE, GIVEN THE PREVALENCE OF THAT DISEASE IN THAT AREA.
Q: IF THE TREATMENT FOR THIS AILMENT IS NOT LICENSED WITHIN THIS COUNTRY, HOW COULD CIVILIANS WHO CONTRACT IT BE TREATED?
BLANCK: THE CENTER FOR DISEASE CONTROL ALSO HAS AN INVESTIGATIONAL NEW DRUG PROTOCOL FOR THE DRUG PENTOSTAM. SO CIVILIANS COULD GET THE TREATMENT THROUGH THE CDC.
Q: ANY NEGATIVE SIDE EFFECTS OF THAT DRUG?

/****** BEGINNING OF SECTION 006 ******/
BLANCK: THERE ARE SOME SIDE EFFECTS THAT PERHAPS COLONEL OSTER COULD SPEAK BETTER TO. ARE THERE ANY OTHER QUESTIONS ON THE BLOOD SUPPLY AT THIS PDINT, AND THEN I'LL TURN IT BACK OVER TO CHUCK.
Q: CAN YOU OFFER ANYTHING MORE DEFINITIVE ON HOW LONG IT WILL TAKE TO DEVELOP A SCREENING? ARE WE TALKING WEEKS OR MONTHS? HOW LONG WILL THIS STAY ON DONATIONS HAVE TO GO BACK?
BLANCK: I DON'T KNOW THE ANSWER TO THAT. OBVIOUSLY, A LOT OF THINGS DEPEND ON THE AVAILABILITY OF A RELIABLE, CONSISTENT TEST. WHILE THE TEST IS FAIRLY STRAIGHT FORWARD, THEN YOU HAVE TO TEST THE TEST AND MAKE SURE THAT IT CAN BE APPLIED. SO I DON'T HAVE A GOOD ANSWER, OTHER THAN IT WILL BE LONGER THAN WEEKS, CERTAINLY.
Q: YOU'RE PROBABLY AWARE THAT THIS ANNOUNCEMENT TODAY IS GOING TO GENERATE A LOT OF INTEREST AMONG RETURNING VETERANS FROM THE WAR. ARE YOU GEARING UP FOR SOME MAJOR INFLUX OF PEOPLE SEEKING THESE KINDS OF TESTS?
BLANCK: FOR AN ASYMPTOMATIC INDIVIDUAL, AS I'VE INDICATED, I DON'T THINK THERE'S THE NECESSITY FOR THE TEST. WE HAVE GEARED UP IN THE SENSE OF BEING PREPARED, YOU BET, FOR LOTS OF QUESTIONS AND PHONE CALLS, AND WE REALLY ARE REFERRING PEOPLE TO THEIR LOCAL PHYSICIAN.

PHYSICIANS WILL BE, OR ARE IN THE PROCESS NOW OF BEING NOTIFIED ABOUT THIS. THEY HAVE BEEN GIVEN NAMES AND NUMBERS TO CALL FOR INDIVIDUALS TO ANSWER QUESTIONS, TOO. FOR EXAMPLE, COLONEL OSTER AND OTHERS IN THE NAVY AND AIR FORCE.

OSTER: GENERALLY THE DRUG IS VERY WELL TOLERATED. I THINK THE MOST COMMON SIDE EFFECT THAT WE'VE SEEN IS MUSICAL SKELETAL ACHES AND PAINS IN OUR PATIENTS, STARTING THE SECOND WEEK IN THERAPY. OCCASIONALLY, BECOMING BOTHERSOME ENOUGH TO REQUIRE A NON-STEROIDAL ANTI-INFLAMMATORY AGENT, AND ON VERY RARE OCCASIONS, STOPPING THE PENTOSTAM TREATMENT A LITTLE BIT EARLY BECAUSE OF THESE SYMPTOMS. ASIDE FROM THAT, THERE REALLY ARE NO OTHER CLINICAL PROBLEMS WITH THE DRUG. WE DO, ON LABORATORY TESTS, SEE SOME ELEVATION IN THE LIVER ENZYMES CONSISTENTLY, WHICH GO AWAY IMMEDIATELY AFTER STOPPING THE DRUG. WE VERY RARELY HAVE SEEN SOME CHANGES IN THE BLOOD PLATELETS GOING DOWN. SO WE MONITOR FOR THOSE CHANGES IN THE BLOOD AND ADJUST DOSE IF NECESSARY. BASICALLY, IT'S A WELL TOLERATED DRUG. THE ONLY PROBLEM WITH IT IS THAT WE DO HAVE TO GIVE IT INTRAVENOUSLY.

Q: THERE IS NO ANTIDOTE TO THIS GOING INTO THE THEATER, THAT YOU COULD HAVE GIVEN TROOPS?

OSTER: NO, WE DON'T HAVE A PROPHYLACTIC TREATMENT FOR LEISHMANIASIS. SOLDIERS WERE VERY CAREFULLY INSTRUCTED ON HOW TO AVOID CONTACT WITH INSECTS, AND THEY DID HAVE REPELLANTS AND WERE INSTRUCTED TO USE REPELLANTS AND USE PROMETHRAN IMPREGNATED NETS AND UNIFORMS SO THAT THERE WAS A LOT OF CONCERN ABOUT ARTHROPOD-BORNE DISEASES GOING INTO THE THEATER, AND I THINK BECAUSE OF THAT WE SAW A VERY, VERY LOW INCIDENCE OF THESE DISEASES. FOR EXAMPLE, SAND FLY FEVER IS A PROBLEM, A VIRUS, THAT WE ANTICIPATED SEEING IN SIGNIFICANT NUMBERS OF OUR TROOPS AND ACTUALLY, IN WORLD WAR II IT SEVERELY INCAPACITATED SOME UNITS BECAUSE OF A HIGH ATTACK RATE. WE SAW NO SAND FLY FEVER, AND I THINK IT'S BECAUSE OF THESE PERSONAL PROTECTIVE MEASURES THAT WE SAW SO LITTLE ARTHROPOD-BORNE DISEASES.

Q: ARE THERE ANY OTHER INFECTIOUS DISEASES THAT CROPPED UP IN TROOPS THAT HAVE COME HOME THAT MAYBE HAVEN'T BEEN PUBLICIZED?

OSTER: NO, THERE HAVEN'T BEEN ANY DISEASES THAT HAVE POPPED UP SINCE THEY'VE COME HOME WITH THE EXCEPTION OF THESE SEVEN CASES OF VISCERAL LEISHMANIASIS. ONE OF THOSE PRESENTED IN THEATER, BUT THE OTHERS ALL PRESENTED AFTER THEIR RETURN HOME.

Q: THERE STILL ARE SOME (INAUDIBLE) DOWN IN FLORIDA.

OSTER: SIR, I'M NOT CONVERSANT WITH THAT PROBLEM, BUT I DO KNOW THERE WERE SKIN TEST CONVERSIONS IN SOME SOLDIERS IN THE 24TH, BUT YOU PROBABLY KNOW MORE ABOUT IT THAN I DO. I DON'T HAVE THAT DATA.

Q: IF SOMEONE HAS A SERIOUS DISEASE UNRELATED TO THIS, AND HAS A TRANSFUSION, GETS THIS IN THE SYSTEM, WHAT IMPACT ON THAT PERSON WILL THIS HAVE?

OSTER: AS I SAID EARLIER, OUR BIGGEST CONCERN WOULD BE WITH THOSE PEOPLE WHO ARE IMMUNOSUPPRESSED. AS YOU CAN IMAGINE, THOSE ARE OFTEN PATIENTS WHO ARE GOING TO BE RECEIVING TRANSFUSIONS. A PERSON, FOR EXAMPLE, WITH A BONE MARROW TRANSPLANT. THEY'RE OFTEN TRANSFUSION DEPENDENT FOR SOME TIME, REQUIRING PLATELETS, TRANSFU-

SIONS, SOMETIMES ON A DAILY BASIS. THOSE ARE THE PATIENTS WHO CANNOT RESIST INFECTION. AIDS PATIENTS ARE ANOTHER GROUP OF PATIENTS THAT CANNOT RESIST INFECTION. SO AN ORGANISM LIKE L. TROPICA WHICH YOU AND I WITH NORMAL IMMUNE SYSTEMS WOULD TOLERATE WITH THIS MILD SELF LIMITED ILLNESS THAT WE'VE DESCRIBED, MAY CAUSE WORSE PROBLEMS IN THE IMMUNOSUPPRESSED. THAT'S SHEER SPECULATION BECAUSE WE HAVEN'T SEEN ANY CASE, BUT THAT'S OBVIOUSLY OF CONCERN TO US. I THINK A HEIGHTENED AWARENESS, AGAIN, OF THIS POTENTIAL PROBLEM AMONG PHYSICIANS THAT HAVE BEEN NOTIFIED -- BOTH CIVILIAN AND MILITARY -- MAY HELP US PICK UP ANY CASES SHOULD THEY OCCUR.
Q: HOW DO WE TREAT THE SOLDIERS THAT HAVE ALREADY BEEN DISCHARGED? YOU BRING THEM BACK TO WALTER REED?
OSTER: WHAT WE WOULD RECOMMEND IS THAT THEY EITHER SEE THEIR LOCAL PHYSICIAN OR TO SEEK TREATMENT IN THE NEAREST VA OR ARMY OR NAVY OR AIR FORCE MEDICAL FACILITY. IF WE THEN SUBSTANTIATE THE DIAGNOSIS, YES, WE WILL CONTINUE TO RECOMMEND THAT THEY COME TO WALTER REED BECAUSE, AS I SAID BEFORE, WE HAVE THE ONLY IND FOR TREATMENT. SO ANY IDENTIFIED CASES WE WILL BRING TO WALTER REED.
HALL: THAT WOULD BE A SERVICE,RELATED PROBLEM, RELATED TO THEIR MILITARY SERVICE THERE.
Q: HAVE ANY OF THE 22 KNOWN CASES DONATED BLOOD BEFORE THEY WERE DIAGNOSED?
OSTER: NO SIR.
Q: ARE WE TALKING ABOUT 22 CASES OR SEVEN? TWENTY-TWO OVERALL AND SEVEN WITH THE MORE SERIOUS MANIFESTATIONS?
OSTER: JUST TO CLARIFY THAT. WE HAD 15 CASES OF WHAT WE EXPECTED, THE SKIN LESIONS. THEN WE HAD SEVEN CASES WHERE WE GOT THE ORGANISM FROM THE BONE MARROW IN PATIENTS WHO PRESENTED WITH A SYSTEMIC ILLNESS -- WITH FEVER AND OTHER COMPLAINTS, BUT HAD NO SKIN LESIONS. IT WAS IN THE WORKUP OF THE FEVER THAT WE DISCOVERED THIS INFECTION IN THE BONE MARROW. SO 22 TOTAL CASES OF LEISHMANIASIS; 15 CUTANEOUS, SEVEN VISCERAL.
Q: WHAT ARE THE PRINCIPAL SYMPTOMS THAT COULD GIVE YOU A CLUE THAT AN INFESTATION HAS OCCURRED?
OSTER: THE EXPERIENCE THAT WE'VE HAD WITH THE SIX SYMPTOMATIC PATIENTS, THE THING I WOULD SAY IS PROBABLY THE SINGLE BEST INDICATOR IS AN UNEXPLAINED FEVER IN A SOLDIER RETURNING FROM THE GULF. THE OTHER THINGS ASSOCIATED WITH THAT WERE WATERY DIARRHEA -- AGAIN, WITHOUT ANY EXPLANATION TO ACCOUNT FOR THE DIARRHEA. IN OTHER WORDS, BACTERIAL AND PROTOZOAN PATHOGENS, WERE LOOKED FOR, AND NOTHING WAS FOUND. AND THEN THE ABDOMINAL PAIN, WHICH WAS SOMETIMES VERY NON-DESCRIPT. DIFFUSE, MIGRATORY, CRAMPY ABDOMINAL

/****** BEGINNING OF SECTION 007 ******/
PAIN WHICH IN A FEW PATIENTS LOCALIZED TO THE LEFT UPPER QUADRANT, WHICH WE THINK REPRESENTS AN ENLARGING SPLEEN, AND AN EXPANDING (CAPSULA) HAS NERVE ENDINGS IN IT SO THAT IF YOU GET ACUTE ENLARGEMENT OF THE SPLEEN YOU CAN GET PAIN FROM THAT.
Q: BOB, ARE YOU STILL DOING THE FREEDOM OF INFORMATION ON THE DESERT STORM FILM? ARE YOU IN CHARGE OF THAT?
HALL: AM I IN CHARGE OF IT?

Q: DID YOU PROVIDE THAT FOOTAGE TO 60 MINUTES...
HALL: NO, WE DID NOT.
Q: HOW DID THEY GET THAT? THAT'S DEPARTMENT OF DEFENSE...
HALL: I DON'T KNOW.
Q: THERE'S NOTHING WRONG...
HALL: IT'S MY UNDERSTANDING THEY GOT IT FROM THE WALL STREET JOURNAL, BUT HOW THE WALL STREET JOURNAL GOT IT, I DO NOT KNOW.
Q: CAN WE GET A COPY OF THAT?
HALL: THERE ARE THREE TAPES WE'VE BEEN ASKED FOR, TWO OF WHICH WE HAVE AND WE SHOULD HAVE THIS AFTERNOON. THOSE ARE THE WINGMAN TAPES. THE OTHER TAPE, I UNDERSTAND, IS GOING TO BECOME AVAILABLE TO US FROM, I THINK, THE WALL STREET JOURNAL, AND WE WILL DO THE USUAL PROCESSING ON IT AND RELEASE IT.
Q: ARE THERE ANY MORE THAT YOU'RE GOING TO RELEASE REGARDING APACHE ATTACKS ON THE IRAQIS?
HALL: WE HAVE RELEASED A NUMBER OF TAPES.
Q: ARE THERE ANY MORE THAT YOU'RE GOING TO RELEASE?
HALL: I THINK THE PEOPLE WHO HAVE ASKED FOR TAPES MAVE BEEN ABLE TO GET WHAT THEY WANTED FROM WHAT WE HAVE.
Q: THEY'LL BE RELEASING TWO OF THOSE TAPES THIS AFTERNOON?
HALL: YES. AS SOON AS WE HAVE THE OTHER AVAILABLE, WE WILL RELEASE IT.
PRESS: THANK YOU.
ADMIN
BT

#3408

NNNN

INQUIRE=DOC16D
ITEM NO=00203773
ENVELOPE
CDSN = LGX933 MCN = 91317/25162 TOR = 913171601
OTTUZYUW RUEKJCS3449 3171601-UUUU--RUEALGX.
ZNR UUUUU
HEADER
O 131601Z NOV 91
FM JOINT STAFF WASHINGTON DC
INFO RUEALGX/SAFE
O 131539Z NOV 91
FM SECDEF WASHINGTON DC//OASD(PA):DPL//
TO AIG 8777
AIG 8798
AIG 8799
ACCT DI-XDWD
BT
CONTROLS
UNCLAS
BODY
SUBJ: PUBLIC AFFAIRS GUIDANCE--LEISHMANIASIS
A. SECDEF WASHINGTON DC//ASD:PA// 130727Z NOV 91
1. REF A IS TRANSCRIPT OF DOD PRESS BRIEFING HELD ON 12 NOV 1991 WHICH DEALS WITH LEISHMANIASIS.
2. THIS MESSAGE PROVIDES PUBLIC AFFAIRS GUIDANCE FOR A POTENTIAL HEALTH CONCERN FOR SERVICEMEMBERS WHO SERVED IN SOUTHWEST ASIA DURING DESERT SHIELD/DESERT STORM, AND TO ALERT ALL PAOS TO TAKE SPECIAL NOTE OF TRANSCRIPT OF DOD PRESS BRIEFING, WHICH PROVIDES COMPREHENSIVE QUESTION-AND-ANSWER DISCUSSION OF LEISHMANIASIS.
3. THE FOLLOWING STATEMENT WAS RELEASED IN WASHINGTON DC AT NOON EST, TUESDAY, NOV 12, 1991: QUOTE: MILITARY PHYSICIANS AND RESEARCHERS HAVE DISCOVERED A DISTINCT MEDICAL SYNDROME IN FOLLOWING UP ON SERVICEMEMBERS RETURNING FROM THE PERSIAN GULF. THESE CASES INVOLVE INFECTION WITH A PARASITE TRANSMITTED FROM THE BITES OF SANDFLIES. ONLY 22 SEVICEMEMBERS OUT OF A HALF-MILLION HAVE BEEN FOUND TO HAVE THE PARASITE KNOWN AS LEISHMANIA. WHILE THIS ORGANISM USUALLY CAUSES AN EASILY TREATED SKIN DISEASE, DOCTORS AT WALTER REED ARMY MEDICAL CENTER AND THE WALTER REED ARMY INSTITUE OF RESEARCH HAVE IDENTIFIED THE INFECTION -- VIA A BONE MARROW CULTURE -- IN SEVEN PATIENTS WHO HAVE NO SKIN LESSONS. THESE PATIENTS HAVE MILD ILLNESS, SOME WITH FEVER AND DIARRHEA. NO CASES INVOLVE LIFE-THREATENING ILLNESS. (PARA) MILITARY MEDICAL PERSONNEL ARE NOW TRYING TO DETERMINE HOW PREVALENT THE DISEASE MAY BE AMONG RETURNING SERVICEMEMBERS. ALTHOUGH DOCTORS BELIEVE THE NUMBER OF CASES IS SMALL, THEY WANT TO ENSURE THAT ALL CASES ARE QUICKLY DETECTED AND TREATED. (PARA) THESE FORMS OF LEISHMANIASIS ARE NOT CONTAGIOUS IN PERSON-TO-PERSON CONTACT ALTHOUGH THERE ARE NO CASES OF THIS SPECIFIC FORM BEING TRANSMITTED THROUGH BLOOD TRANSFUSIONS, FIVE CASES OF A RELATED STRAIN ARE REPORTED IN THE MEDICAL LITERATURE TO HAVE BEEN TRANSMITTED BY TRANSFUSION OF CONTAMINATED BLOOD. ALTHOUGH THE RISK OF CONTAMINATION IS VERY LOW, DOD, IN THE INTEREST OF SAFETY, WILL RECOMMEND THAT ALL

PEOPLE WHO HAVE TRAVELLED TO THE GULF AREA, (INCLUDING SAUDI ARABIA, KUWAIT, IRAQ, BAHRAIN, QATAR, THE UNITED ARAB EMIRATES, OMAN AND YEMEN), SINCE AUG 1, 1990, TEMPORARILY REFRAIN FROM DONATING BLOOD. THIS DELAY WILL ENABLE MEDICAL RESEARCHERS TO DETERMINE THE LEVEL OF ADDITIONAL INFECTIONS AMONG THE EXPOSED POPULATION AND WILL ALSO ALLOW THEM TO DEVELOP A SCREENING TEST FOR INFECTION. (PARA) IN ADDITION, DEPARTMENT OF DEFENSE BLOOD PROGRAM OFFICIALS ARE WORKING CLOSELY WITH THE FOOD AND DRUG ADMINISTRATION, THE CENTERS FOR DISEASE CONTROL, THE AMERICAN RED CROSS, THE AMERICAN ASSOCIATION OF COUNCIL OF COMMUNITY BLOOD CENTERS, AND OTHER GROUPS INVOLVED WITH THE COUNTRY'S SUPPLY OF BLOOD AND BLOOD PRODUCTS, TO DECIDE HOW BEST TO HANDLE THE SITUATION. UNQUOTE

4. THE FOLLOWING QUESTIONS AND ANSWERS ARE PROVIDED:

Q1: WHAT ARE THE SYMPTOMS?

A1: THE ORGANISM USUALLY CAUSES A SKIN LESION, BUT MAY ALSO INCLUDE UNEXPLAINED FEVER, DIARRHEA, AND ABDOMINAL PAIN.

Q2: HOW IS LEISHMANIASIS TREATED?

A2: TREATMENT USUALLY CONSISTS OF ADMINISTERING A DRUG CALLED PEN, TOSTAM FOR 30 DAYS.

Q3: ONCE TREATED, IS THE SICKNESS CURED, OR CAN THE PARASITE RE-EMERGE, AS WITH MALARIA?

A3: IT CAN BE EFFECTIVELY TREATED; WE DO NOT EXPECT SYMPTOMS TO RECUR, BUT THERE IS A POTENTIAL FOR RELAPSE WHEN A PERSON DEVELOPS AN IMMUNE DEFICIENCY (AS IN THE CASE OF A PERSON WHO HAS DEVELPOED AIDS OR HAS HAD AN ORGAN TRANSPLANT AND MUST TAKE AN IMMUNOSUPPRESSION DRUG).

Q4: HAS THERE BEEN ANY CASE OF AN IDENTIFIED INFECTED PERSON DONATING BLOOD?

A4: NO

Q5: HOW MANY SERVICEMEMBERS SERVED IN SOUTHWEST ASIA SINCE AUGUST 1, 1990?

A5: 541,425

Q6: ARE INFECTED SERVICEMEMBERS A RISK TO THOSE WITH WHOM THEY LIVE AND WORK?

A6: PERSON-TO-PERSON TRANSMISSION OF THIS PARASITE HAS NOT BEEN REPORTED.

Q7: WHAT ARE THE MOST SEVERE CONSEQUENCES OF HAVING THIS PARASITE?

A7: THIS ILLNESS IS NOT FATAL, AND IT IS SELF-LIMITING, WHICH MEANS THAT IT WILL NOT PROGRESS TO MORE SERIOUS ILLNESS. SKIN LESIONS, HIGH FEVER, AND DIARRHEA ARE THE ONLY KNOWN DIFFICULTIES.

Q8: WHAT CAN SERVICEMEMBERS DO TO FIND OUT WHETHER THEY HAVE THE PARASITE?

A8: ANYONE WHO EXPERIENCES ANY OF THESE SYMPTOMS SHOULD REPORT TO A MEDICAL CLINIC FOR EVALUATION. THE MEDICAL OFFICER WILL DETERMINE IF SPECIALIZED TREATMENT IS REQUIRED.

Q9: WHERE CAN A VETERAN GO TO GET A BLOOD TEST?

A9: THERE IS NO COMMERCIALLY AVAILABLE BLOOD TEST FOR THIS SYNDROME AT THIS TIME. WE HAVE DISTRIBUTED INFORMATION VIA A "DEAR COLLEAGUE" LETTER TO MEDICAL PERSONNEL. WE HAVE ALSO PROVIDED NAMES AND ADDRESSES OF MEDICAL PERSONNEL WHO ARE AVAILABLE FOR CONSULTATION ON THIS MATTER. THIS INFORMATION COMPLEMENTS THE ARTICLES PUBLISHED BY

MILITARY DOCTORS IN THE NEW ENGLAND JOURNAL OF INFECTIOUS DISEASE TO ALERT THE MEDICAL COMMUNITY OF THE POSSIBILITY OF SUCH INFECTIONS.
Q10: WHEN DID YOU FIND OUT ABOUT THE PROBLEM?
A10: THE PROBLEM WAS BROUGHT TO THE ATTENTION OF DOD HEALTH AFFAIRS OFFICIALS LAST WEEK. THEY MADE A DECISION ON FRIDAY, NOV. 8, TO SUSPEND BLOOD DONATIONS FROM ALL PERSONNEL WHO TRAVELLED TO THE GULF AREA.
5. THE OASD(PA) POC FOR PLANS IS MAJ STEVE LITTLE, USMC, AT DSN 223-1075, COMM (703) 693-1075; POC FOR MEDIA QUERIES IS SUSAN HANSEN, AT DSN 223-0192, COMM (703) 695,0192.
ADMIN
BT

#3449

NNNN

INQUIRE=DOC13D
ITEM NO=00718981
ENVELOPE
CDSN = LGX120 MCN = 91061/18710 TOR = 910611647
RTTUZYUW RUEKJCS8891 0611645-UUUU--RUEALGX.
ZNR UUUUU
HEADER
R 021645Z MAR 91
FM JOINT STAFF WASHINGTON DC
INFO RUEALGX/SAFE
R 021640Z MAR 91 ZNZ1
FM FM SECDEF WASHINGTON DC//ASD:PA//
TO AIG 8798
AIG 8799
RXFBI/AFCENT BRUNSSUM NL
ACCT DA-BHCWAA
BT
CONTROLS
UNCLAS , NATO UNCLASSIFIED FOR NATO ADDRESSEES
SECTION 01 OF 08
FOR PUBLIC AFFAIRS OFFICERS

/********** THIS IS A COMBINED MESSAGE **********/
BODY
SUBJECT: DOD NEWS BRIEFING
DELIVER DURING NORMAL DUTY HOURS
FOLLOWING IS THE TRANSCRIPT OF A NEWS BRIEFING CONDUCTED BY MR. PETE WILLIAMS, ASD/PUBLIC AFFAIRS; LT GEN THOMAS KELLY, USA; REAR ADMIRAL MIKE MCCONNELL, USN, AT THE PENTAGON, ON FRIDAY, MARCH 1, 1991 AT 3 P.M.:
MR. WILLIAMS: GOOD AFTERNOON. GENERAL TOM KELLY, DIRECTOR OF OPERATIONS FOR THE JOINT STAFF; AND REAR ADMIRAL MIKE MCCONNELL, DIRECTOR OF INTELLIGENCE FOR THE JOINT STAFF, ARE OUR BRIEFERS TODAY. GENERAL KELLY WILL MAKE HIS USUAL OPENING STATEMENT, AND THEN THEY'LL TAKE YOUR QUESTIONS. I DON'T BELIEVE ADMIRAL MCCON, NELL HAS ANY PREPARED BUSINESS FOR YOU TODAY.
AT THE CONCLUSION OF THE BRIEFING WE WILL BE DISTRIBUTING THE SECRETARY'S ANNUAL REPORT TO THE PRESIDENT AND CONGRESS, WHICH, AS YOU KNOW, IS REQUIRED BY LAW ONCE A YEAR. THE DEPARTMENT IS REQUIRED TO REPORT TO THE WHITE HOUSE AND THE CONGRESS ON ITS ACTIVITIES OF THE PREVIOUS YEAR AND ITS PLANS FOR THE FORTHCOMING YEAR. THE PUBLICATION IS BEING SENT TO THE PRESIDENT AND THE CONGRESS TODAY, SO THAT WILL BE AVAILABLE DOWN IN OUR DIRECTORATE FOR DEFENSE INFORMATION AT THE END OF THIS BRIEFING.
YOU'VE HEARD THE PRESIDENT SAY THAT THE MEETING TOMORROW BETWEEN THE UNITED STATES AND THE COALITION FORCES, MEETING WITH THE REPRESENTATIVES OF IRAQ ,, WHOEVER THEY MAY BE ,, WILL TAKE PLACE TOMORROW. WE DON'T HAVE ANY FURTHER INFORMATION ON THAT HERE. WE DON'T HAVE A PRECISE TIME FOR YOU, AND I DON'T BELIEVE THERE WILL BE ANY ANNOUNCEMENT OF THE PRECISE LOCATION BEFORE THE MEETING TAKES PLACE. OBVIOUSLY, THE IRAQIS HAVE TO BE NOTIFIED

OF TH'S, WE UNDERSTAND THAT, AND WE WILL MAKE ARRANGEMENTS, AND IN FACT ARE WORKING ON THOSE ARRANGEMENTS NOW WITH YOUR PARENT NEWS ORGANIZATIONS TO ARRANGE FOR PRESS COVERAGE, INTERNATIONAL PRESS COVERAGE OF THIS EVENT. SO ALL OF THE MAJOR NEWS ORGANIZA, TIONS WILL BE REPRESENTED ONE WAY OR ANOTHER THERE. WE'RE TRYING TO WORK THAT OUT WITH THE NETWORKS AND THE NEWSPAPERS AND THE WIRES AND THE MAGAZINES AND RADIO NETWORKS AND SO FORTH RIGHT NOW. WE WILL HAVE MORE TO SAY ABOUT THAT TO YOUR ORGANIZATIONS. BUT WE DON'T HAVE ANY FURTHER INFORMATION HERE ON WHEN OR WHERE THAT WILL BE HELD OR THE ARRANGEMENTS FOR IT, BUT WE ARE WORKING ON COVERAGE.

WITH THAT, LET ME INTRODUCE GENERAL KELLY AND ADMIRAL MCCONNELL.

GENERAL KELLY: THANKS, PETE.

GOOD AFTERNOON. I'D LIKE TO START TODAY WITH THE OVERALL SITUA, TION IN THE THEATER. THE ARRANGEMENTS ARE ONGOING TO SET UP THE MEETING BETWEEN IRAQI AND COALITION MILITARY LEADERS. NO FORMAL CEASEFIRE WILL EXIST UNTIL ALL COALITION DEMANDS ARE MET. U.S. FORCES ARE IN DEFENSIVE POSITIONS, READY TO RESUME THE OFFENSIVE IF REQUIRED. SOME MINOR REPOSITIONING OF THOSE FORCES IS TAKING PLACE. KUWAIT INTERNATIONAL AIRPORT IS OPEN FOR C,130'S FLYING IN SUPPLIES.

RECONNAISSANCE INDICATES NUMEROUS CONVOYS ,- SOME AS LARGE AS 60 VEHICLES ,- HEADED TOWARDS BAGHDAD. THESE ARE REMNANTS OF THE FORCES THAT ESCAPED TO THE BASRA AREA. NO ADDITIONAL FORCES HAVE BEEN SEEN LEAVING THE KUWAITI THEATER OF OPERATIONS.

PERSONNEL REMAINS THE SAME AT ABOUT 539,000. CASUALTIES HAVE GONE UP TEN SINCE YESTERDAY, AND THERE IS NORMALLY A REPORTING LAG FROM THE FIELD ON THINGS LIKE THAT, SO IT'S NOT UNUSUAL. KILLED IN ACTION PRIOR TO THE GROUND WAR, 23; SINCE THE GROUND WAR BEGAN, 38 -, 10 MORE THAN YESTERDAY; IN THE SCUD ATTACK, 28, FOR A TOTAL OF 89 KILLED IN ACTION. WOUNDED PRIOR TO THE GROUND WAR, 34; SINCE THE GROUND WAR, 78; WOUNDED AS A RESULT OF THAT SCUD ATTACK, 212; MISSING IN ACTION, 38; POW'S, NINE. THERE HAVE BEEN NO MORE SCUDS FIRED.

THE ENEMY PRISONER OF WAR NUMBERS ARE DYNAMIC, AND THE BEST WE HAVE YET IS STILL ABOVE 50,000. TURKEY HAS ABOUT 3147.

IRAQI GROUND EQUIPMENT DESTROYED, WE HAVE THE SAME NUMBERS THAT WE HAD YESTERDAY, 3008 TANKS; 1856 ARMORED VEHICLES; 2140 ARTIL-LERY PIECES. MORE REPORTS ARE COMING IN, BUT THEY NEED TO BE COLLATED. WHEN I SAY COMING IN, I MEAN COMING INTO THEATER HEADQUARTERS THERE. BUT FOR EXAMPLE, THE MARINES REPORTED DESTROYED OR CAPTURED 1060 TANKS; 608 APC'S; 432 ARTILLERY PIECES; FIVE FROGS; AND TWO TELS (TRANSPORTER ERECTOR LAUNCHERS) FOR THE SCUDS.

MEDICAL, 1850 PATIENTS ARE IN U.S. HOSPITALS. MOST ARE SUFFERING FROM ROUTINE ILLNESSES. 170 ARE ENEMY PRISONERS OF WAR ,, 80 PERCENT OF THEM ARE COMBAT INJURIES. THE REST ARE SUFFERING FROM MALNUTRITION AND DEHYDRATION.

IN TERMS OF THE GROUND FORCES OPERATIONS, U.S. FORCES ARE STILL CLEARING OBSTACLES AND TAKING CARE OF SMALL POCKETS OF IRAQI RESISTANCE AS THEY ARISE. THERE'S NO CONTINUOUS THING GOING ON

OVER THERE.
THERE'S OBVIOUSLY, STILL NO INFORMATION FROM THE IRAQIS ON THE LOCATIDN OF THE MINES, BECAUSE THEY HAVEN'T MET YET.
AIR OPERATIONS REMAIN DEFENSIVE IN NATURE. DEFENSIVE COUNTER, AIR, RECONNAISSANCE, SCUD PATROL, AND RESUPPLY. SORTIES ARE OVER 110,000.
WE HAD A LEFTOVER QUESTION FROM YESTERDAY, HAVE YOU DISCOVERED THE WRECKAGE OF THE AC-130. THE ANSWER IS NO, WE HAVE SEARCHED FOR IT AND HAVE NOT YET FOUND IT; ERGO, WE HAVE NO INFORMATION ON THE DISPOSITION ON THE GROUND CREW. ONE WOULD HOPE THEY WERE PRISONERS AND NOT LOST.
Q: GENERAL KELLY, HOW IMPORTANT ARE THESE AMERICAN PRISONERS OF WAR TO YOU IN THE TALKS THAT ARE GOING TO HAPPEN TOMORROW? WHAT HAS TO HAPPEN AT THOSE TALKS ABOUT THOSE PEOPLE?
KELLY: I THINK THE LEADERSHIP HAS BEEN VERY CONSISTENT IN WHAT THEY SAID. THE TOP PRIORITY IS TO GET THE PRISONERS BACK -- NOT ONLY OUR PRISONERS OF WAR, BUT THE COALITION PRISONERS OF WAR, CIVILIAN HOSTAGES, THE CBS NEWS CREW. IN MY VIEW, WE'RE DEALING WITH HUMANITY HERE, SO ALL OF THEM WOULD HAVE EQUAL PRIORITY. I THINK IT'S GOING TO BE MADE VERY CLEAR, UNLESS THE IRAQI FORCES ARE WILLING TO DEAL ON THAT ISSUE FIRST, THAT WE'RE NOT GOING TO GET VERY MUCH FURTHER. AS I SAID, WE'RE PREPARED TO RESUME OPERATIONS SHOULD THAT BECOME NECESSARY.
Q: WHAT DOES THAT MEAN, SIR? WHAT DOES PREPARED TO RESUME OPERATIONS MEAN? IF THEY SAY NOTHING ABOUT THE POW'S TOMORROW, YOU'RE GOING TO START ATTACKING AGAIN?
KELLY: NO, IT MEANS IF THEY SAY NOTHING ABOUT THE POW'S TOMORROW THAT THE PRESIDENT WILL BE INFORMED OF THAT, AND HE WILL MAKE A DECISION, AND WE ARE PREPARED TO EXECUTE ANY DECISION THAT HE MAKES.
Q: COULD YOU GIVE US A SENSE OF WHAT ELSE WILL BE DISCUSSED AT THE MEETING TOMORROW?
KELLY: YES, AS A MATTER OF FACT, THE PRESIDENT LAID IT OUT

/****** BEGINNING OF SECTION 002 ******/
PRETTY CLEARLY YESTERDAY. THEY HAVE TO AGREE TO THE UN RESOLUTIONS. THEY HAVE TO RETURN ALL OF THE PRISONERS THAT THEY HOLD EITHER AS HOSTAGES OR AS PRISONERS OF WAR. AS A MATTER OF FACT, THE FIRST THING, "RELEASE IMMEDIATELY ALL COALITION PRISONERS OF WAR, THIRD COUNTRY NATIONALS, AND THE REMAINS OF ALL WHO HAVE FALLEN. INFORM THE KUWAITI AUTHORITIES OF THE LOCATION AND NATURE OF ALL LAND AND SEA MINES. COMPLY FULLY WITH RELEVANT UN SECURITY COUNCIL RESOLUTIONS INCLUDING A RESCINDING OF IRAQ'S AUGUST DECISION TO ANNEX KUWAIT. AND THE ACCEPTANCE IN PRINCIPLE OF RESPONSIBILITY TO PAY COMPENSATION FOR THE LOSS, DAMAGE, AND INJURY THIS AGGRESSION HAS CAUSED. DESIGNATE MILITARY COMMANDERS TO MEET WITH OURS. A CEASEFIRE IS CONTINGENT UPON IRAQ NOT FIRING UPON ANY COALITION FORCES, AND NOT LAUNCHING ANY SCUD MISSILES AGAINST ANY OTHER COUNTRY. IF IRAQ VIOLATES THESE TERMS, COALITION FORCES WILL BE FREE TO RESUME MILITARY OPERATIONS."

Q: YOU MENTIONED THE MINES. THERE ARE SO MANY MINES OUT IN THE SEA; AND EVEN IF THEY TELL YOU, WHAT KIND OF A JOB IS IT GOING TO TAKE TO CLEAR THOSE MINES ,- BOTH LAND AND SEA? DO YOU HAVE ANY SENSE NOW THAT THERE WAS A PATTERN TO THE WAY THE MINES WERE PUT OUT?

KELLY: THE FIRST ANSWER IS, IT'S A VERY BIG JOB. THE SECOND ANSWER IS YES, THEY WERE PUT OUT IN PATTERNS, BUT SOME OF THE MINES BROKE LOOSE. RIGHT NOW, IF THEY DON'T TELL US WHAT THE PATTERN WAS, WE'VE GOT TO WORK THAT OUT FOR OURSELVES, WHICH MAKES THE JOB EXPONENTIALLY MORE DIFFICULT, BUT WE HAVE HAD BIG MINE CLEARING JOBS BEFORE ,, REMEMBER THE SUEZ CANAL HAD A LOT OF MINES IN IT AND OTHER PLACES. WE'RE PRETTY GOOD AT THAT, BUT IT'S GOING TO TAKE THE COALITION A LONG TIME TO GET ALL THAT CLEARED. THAT'S THE SEA MINES. YOU STILL HAVE A PROBLEM WITH THE LAND MINES, SOME OF WHICH ARE SEEDED, THE ANTI-TANK MINES ARE SEEDED WITH ANTI-PERSONNEL MILES, AND IT'S A TOUGH JOB TO GET THEM OUT.

Q: YOU SAID TODAY THAT YOU DIDN'T HAVE ANY MORE INFORMATION ON THIS AC,130 GUNSHIP WHICH WENT DOWN. THE C-130 PLANE WAS THE LARGEST PLANE IN THIS ENTIRE OPERATION TO GO DOWN, IF I'M NOT CORRECT, IN THE THEATER. WE HAVE SATELLITES, YOU HAVE A LOT OF OPTIONS TO SEE SMALL THINGS, AS SMALL AS A TEMPLE. DO YOU MEAN TO TELL ME THAT YOU HAVE NO INFORMATION TO GIVE US ON WHERE THIS PLANE IS, WE DON'T EVEN KNOW IF IT WENT DOWN IN THE WATER OR THE LAND? CAN'T YOU TELL US SOMETHING, OR YOU WILL NOT TELL US SOMETHING BECAUSE IT WAS A SPECIAL OPERATION AIRPLANE?

KELLY: WE DON'T HAVE THE INFORMATION. IF I WAS WITHHOLDING IT BECAUSE IT WAS A SPECIAL OPS AIRPLANE, I'D TELL YOU. IT WAS A SPECIAL OPS AIRPLANE, BY THE WAY, AS YDU KNOW. THAT'S A BIG AIRPLANE, BUT BIG IS RELATIVE. IRAQ IS 90,000 SQUARE MILES. IT COULD HAVE GONE INTO (AL WADI), IT COULD HAVE GONE INTO WATER. WE'RE LOOKING FOR IT. WE CARE ABOUT THOSE FOLKS, AND WE'RE GOING TO DO EVERYTHING WE CAN TO FIND IT, BUT WE HAVE NOT FOUND IT YET.

MCCONNELL: OUR INTELLIGENCE SYSTEMS ARE INCREDIBLE, THEY'RE WONDERFUL, AND THEY DID A MAGNIFICENT JOB IN THIS EFFORT. BUT WE CAN'T SEE EVERYTHING ALL THE TIME. YOU ASK IF WE CAN SEE A TEMPLE, WHY CAN'T WE FIND SOMETHING AS LARGE AS A LARGE AIRPLANE -, A TEMPLE DOESN'T MOVE. IF YOU'RE LOOKING FOR SOMETHING THAT YOU DON'T KNOW WHERE TO START YOUR SEARCH, IT BECOMES OFTEN A NEEDLE IN A HAYSTACK, AND THE EXAMPLE IS A MOBILE SCUD.

Q: IF A CIVILIAN AIRCRAFT CAN HAVE A BLACK BOX AND CAN BE FOUND IN THE INDIAN OCEAN OR WHATEVER, I'M SURE THE AIR FORCE HAS A HIGHER CAPABILITY TO FIND SOMETHING LIKE THIS. AS A FOLLOWUP TO THAT, ON THE AGENDA FOR TOMORROW, WILL THERE BE DISCUSSIONS OF MIA'S AND THIS AIRPLANE, WHICH IS A MASSIVE THING THAT'S SOME, WHERE IN KUWAIT?

KELLY: HONEST TO GOD, WE'RE TRYING TO FIND THE AIRPLANE. WE'RE USING EVERY SYSTEM WE HAVE TO DO IT. WE HAVE ALREADY SAID THAT THE ISSUE OF PERSONS WHO ARE MISSING IN ACTION WILL BE THE TOP AGENDA ITEM FOR THE MEETING TOMORROW.

Q: DID YOU TAKE ANY MILITARY ACTIONS BEFORE THE WAR ENDED TO SEE

IF YOU COULD PREVENT SADDAM HUSSEIN FROM GETTING OUT OF IRAQ? WHAT ARE YOU DOING NOW THAT THE WAR IS OVER, TO MAKE SURE THAT HE DOES NOT FLEE TO SOME SANCTUARY?
KELLY: BEFORE THE WAR ENDED, WE STRUCK NORMAL TARGETS, AS I THINK WAS ANNOUNCED, SOME OF THEM HAPPENED TO BE CIVILIAN JETS THAT WERE LOCATED AT THE AIRPORT IN BAGHDAD. THAT'S ABOUT THE EXTENT OF ANYTHING THAT COULD BE CONSTRUED AS A PREVENTIVE MEASURE FROM TRYING TO GET HIM OUT OF IRAQ. CAN YOU THINK OF ANYTHING ELSE, MIKE?
MCCONNELL: THERE WAS NO DELIBERATE TARGETING OF THE MAN HIMSELF, AND THERE'S BEEN NO FOLLOWUP SINCE THEN.
Q: WHAT ARE YOU DOING NOW, IF ANYTHING, TO MAKE SURE HE DOESN'T FLEE?
KELLY: WE'RE FLYING DEFENSIVE AIR CAPS. THAT IS NOT SPECIFICALLY TO GET MR. HUSSEIN IF HE'S TRYING TO LEAVE THE COUNTRY. HOWEVER, I'M SURE IF AN AIRPLANE TOOK OFF AND THERE WAS ANY INDICATION THAT HE WAS ON IT, THE MILITARY LEADERSHIP IN THEATER WOULD CALL BACK IMMEDIATELY FOR INSTRUCTIONS AND A DECISION WILL BE MADE. I DON'T KNOW OF A DECISION THAT'S PRE,POSITIONED THAT SAYS STOP SADDAM HUSSEIN FROM LEAVING BAGHDAD.
Q: YOU HAVE THIS COMBAT AIR PATROL OVER ALL OF IRAQ SO THAT IF HE DOES SOMEHOW GET TO A NORTHERN AIRFIELD...
KELLY: WE HAVE IT OVER LARGE PORTIONS OF IRAQ. WE, OBVIOUSLY, HAVE IT OUT IN THE WESTERN SCUD BASKET BECAUSE WE'RE STILL VERY CONCERNED ABOUT THAT, AND WE ARE FLYING COMBAT AIR PATROLS OVER BAGHDAD.
Q: THERE'S A REPORT THAT THE IRAQI COMMANDERS IN KUWAIT CITY FLED BEFORE THEIR TROOPS AND ELUDED THE ALLIES; AND THAT THE COMMANDER OF THE KUWAITI THEATER OF OPERATIONS FLED AS WELL, AND SUPPOSEDLY WAS THE ONE WHO USED THE CHEMICAL WEAPONS ON THE KURDS. I WONDER IF YOU HAVE INFORMATION TO CONFIRM THIS, AND WHAT YOUR JUDGMENT WOULD BE ABOUT THAT, IF IT DID HAPPEN?
KELLY: IF A COMMANDER FLED HIS TROOPS, THAT'S SCURRILOUS. WE'VE SEEN THAT BEFORE. WE SAW IT IN PANAMA, BY THE WAY, WITH SOME OF THE LEADERSHIP DOWN THERE. DECENT LEADERS HANG AROUND WHEN IT GETS TOUGH.
MCCONNELL: I'M AWARE OF THE REPORTS, BUT THERE'S NO WAY WE HAVE CORROBORATING INFORMATION ON ANY OF THAT.
Q: HAVE ANY OF YOUR INTELLIGENCE TEAMS FOUND QUANTITIES OF CHEMICAL MUNITIONS, WHAT TYPES, AND ANY BIOLOGICAL MUNITIONS?
KELLY: WE HAVE FOUND NO CHEMICAL MUNITIONS YET, TO MY KNOWLEDGE. I BELIEVE IT WAS ANNOUNCED IN,THEATER TODAY THAT THEY HAD FOUND A CHEMICAL BUNKER AND THEY WERE EXPLOITING IT, DID NOT FURTHER IDENTIFY WHERE THE BUNKER WAS, DID NOT IDENTIFY WHAT WAS IN IT. WE HAD FOUND A BUNKER PREVIOUSLY, AS YOU KNOW, THAT HAD SOME

/****** BEGINNING OF SECTION 003 ******/
CHEMICAL MARKINGS ON IT, BUT THERE WERE NO CHEMICALS IN IT. EPW REPORTS INDICATE THAT THE IRAQI MILITARY FORCES WERE VERY UNCOMFORTABLE WITH THE USE OF CHEMICAL WEAPONS. AS A MATTER OF FACT, MANY OF THEM HAD NO CHEMICAL PROTECTIVE CAPABILITY.

Q: WAS THE BUNKER IN THE KTO OR WAS IT ON THE OTHER SIDE OF THE IRAQ BORDER?
KELLY: THEY DIDN'T SPECIFY WHERE IT WAS, SO I DON'T KNOW. I THINK THEY WERE CAREFUL NOT TO SPECIFY WHERE IT WAS. I FEEL SURE IT WAS IN THE KTO, BECAUSE THAT'S WHERE OUR FORCES ARE ,- THEY'RE NOT NORTH OF THERE. BUT I DON'T KNOW IF IT WAS IN IRAQ OR KUWAIT.
Q: WHAT IS OUR VIEW TOWARD THE EQUIPMENT THAT MAY NOT HAVE BEEN HEAVILY DAMAGED, AND CERTAINLY NOT DESTROYED, THAT WAS LEFT BEHIND BY THE FLEEING IRAQIS IN KUWAIT? IN TWO RESPECTS, NUMBER ONE, TO WHOM DOES IT BELONG; AND NUMBER TWO, ARE WE ATTEMPTING TO DESTROY IT EVEN DURING THIS PERIOD OF THE SUSPENSION OF OFFENSIVE OPERATIONS?
KELLY: SOME OF THE STUFF THAT WAS FOUND ON THE BATTLEFIELD WAS BEING DESTROYED, AND YOU SAW TV PICTURES OF THAT ,- THERMITE GRENADES IN THE TUBE, THERMITE GRENADES IN THE TURRET. OTHERS WILL PROBABLY NOT BE DESTROYED. IT BELONGS TO US -- IT'S A SPOIL OF WAR, I'M SURE. IT'S NOT GOING TO BE GIVEN BACK, CERTAINLY, TO THE IRAQIS. WHETHER OR NOT WE WOULD DEVELOP PLANS FOR THE FUTURE USE OF THAT EQUIPMENT ,- WHEN I SAY WE, I MEAN THE COALITION -- I DON'T KNOW. THAT DECISION HASN'T BEEN MADE YET, TO MY KNOWLEDGE.
Q: DOES THE SEARCH FOR SCUDS, IS THAT LIMITED SOLELY TO AIR RECONNAISSANCE? ARE THERE ANY GRDUND OPERATIONS INVOLVED IN WESTERN IRAQ OR ELSEWHERE LOOKING FOR SCUDS?
KELLY: RIGHT NOW THE ONLY THING WE HAVE LOOKING FOR SCUDS IN WESTERN IRAQ ARE THE AIRPLANES, THAT WE'LL TALK ABOUT.
Q: EXCUSE ME, WHAT WAS THAT LAST?
A: THAT WE'LL TALK ABOUT.
Q: IS IT TRUE THAT PRESIDENT BUSH HAS ORDERED THE JOINT CHIEFS TO DEVELOP AN ESTIMATE OF IRAQI CASUALTIES? AND WHY HAS THE PENTAGON REMAINED SO RELUCTANT, EVEN AFTER THE FIGHTING ENDED, TO DD THAT?
KELLY: TO THE BEST OF MY KNOWLEDGE, THERE HAS BEEN NO DIRECTIVE GIVEN TO THE JOINT CHIEFS OR ANYONE ELSE TO DEVELOP AN ESTIMATE OF IRAQI CASUALTIES. IRAQ SHOULD BE CAPABLE OF DOING THAT, BY THE WAY. IT IS EXTREMELY DIFFICULT FOR US TO DO BECAUSE, AS YOU MAY KNOW, WE'VE HAD EPW REPORTS, AND THEY DID THIS IN THE IRAN/IRAQ WAR, THEIR TENDENCY IS, TO KEEP MORALE UP, TO BURY THE DEAD WHILE IT'S STILL DARK. SO THERE MAY BE HIDDEN MASS GRAVES, OR MASS GRAVES HIDDEN OR NOT, UP THERE THAT WE DON'T EVEN KNOW ABOUT. AS I SAY, WE DON'T HAVE ANY GUIDANCE THAT WE NEED TO COME UP WI H A NUMBER. THE NUMBER THAT I CAN COME UP WITH YOU IS ONE IRAQI ARMY DESTROYED. (LAUGHTER)
Q: CLEARLY, SADDAM HUSSEIN HAS NO INTEREST IN DEVELOPING SUCH A NUMBER, IF IT'S A HUGE NUMBER. AND CERTAINLY, WHILE IT WOULD BE A GRUESOME NUMBER, WE WOULD HAVE SOME INTEREST IN GETTING A NUMBER OUT THERE. GENERAL NEAL SAID THIS MORNING HE THOUGHT WHEN THE IRAQI PEOPLE REALIZED HOW GREAT THE CASUALTIES HAD BEEN, THAT WOULD BE ONE OF THE THINGS THAT WOULD MAKE THEM WANT TO GET RID OF SADDAM HUSSEIN.
KELLY: I'LL STICK WITH THE ANSWER I GAVE YOU. I THINK THAT THE

IRAQI PEOPLE WILL KNOW OF THE MAGNITUDE OF THAT NUMBER A COUPLE OF DIFFERENT WAYS. NUMBER ONE, SOLDIERS NOT COMING HOME; NUMBER TWO, RIGHT NOW WE SEE LARGE NUMBERS OF IRAQI SOLDIERS WHO HAVE BEEN ABLE TO CROSS INTO IRAQ AND ARE HEADING NORTH TOWARDS BAGH, DAD. WHEN THEY GET BACK AND TELL THE STORIES OF WHAT THEY WERE REQUIRED TO FACE AND THE RESULTS OF THE IMPERIAL CAMPAIGN AS PURSUED BY THE LEADERSHIP IN BAGHDAD, I THINK WHAT GENERAL NEAL INDICATED MIGHT BE POSSIBLE IS GOING TO HAPPEN. THAT'S MY OPIN, ION.

Q: IT'S BEEN SAID, ACTUALLY SEVERAL TIMES IN THE PENTAGON, THAT THE UNITED STATES HAS WON THE WAR SEVERAL TIMES, AND THERE'S BEEN AN ABSOLUTE VICTORY, BUT THAT THE PEACE IS DIFFICULT. AS A MILITARY MAN, WHAT DO YOU WANT TO SEE THAT WILL WIN THE PEACE ,, PARTICULARLY IN THE TALKS TOMORROW, BUT ALSO DOWN THE ROAD?

KELLY: WISE DECISIONS, EFFECTIVE IMPLEMENTATION OF THOSE WISE DECISIONS. (LAUGHTER) IN SOME WAYS YOU CAN SAY THE EASY PART'S OVER. PRESIDENT BUSH ALLUDED TO THAT -, NOT THAT THE EASY PART'S OVER, BUT ALLUDED TO WINNING THE PEACE. MAKING THE PROPER MIX OF DECISIONS, WHICH IS GOING TO BE EXTREMELY DIFFICULT. IT'S A REGION THAT'S BEEN IN TURMOIL FOR MANY, MANY YEARS. THERE ARE A LOT OF CONFLICTING, DYNAMICS THAT ARE PULLING AND PUSHING AGAINST THE BODY OF THE ARAB STATES. SO WE'RE GOING TO HAVE TO BE EXTREMELY CAREFUL -- WE, THE UNITED STATES. THIS IS NOT AN OPERATIONS OFFICER'S JOB, I AM VERY HAPPY TO TELL YOU, BUT WE, THE UNITED STATES, ARE GOING TO HAVE TO BE CAREFUL, ALONG WITH THE OTHER MEMBERS OF THE COALITION, THAT WE DO THE BEST WE CAN IN ORDER TO TRY TO PUT SOME PEACE AND STABILITY IN THE AREA. YOU'LL RECALL AFTER WORLD WAR II, WE HAD THE SAME KIND OF DILEMMA. I THINK WE WERE STUNNINGLY SUCCESSFUL, GIVEN THAT THE TWO MAJOR ENEMIES WE HAVE ARE NOW ALLIES. I THINK AMERICANS ALWAYS HAVE A TENDENCY IN VICTORY TO TRY TO WIN OVER THE FORCE THAT'S BEEN DEFEATED. I THINK THAT'S WHAT'S...

Q: SPECIFICALLY IN THE MEETING TOMORROW AND LOOKING DOWN THE ROAD...

KELLY: THAT ONE WE'LL NOT BE ADDRESSING, WHAT YOU'RE TALKING ABOUT. THE MEETING TOMORROW I THINK IS GOING TO BE ADDRESSING SPECIFICALLY WHAT I MENTIONED. AND THE PRESIDENT SAID IN HIS TALK EARLIER TODAY THAT THE SECRETARY OF STATE WAS LEAVING TQDAY TO GO AROUND TO VARIOUS CAPITOLS TO BEGIN TO TALK ABOUT THE DIMENSIONS OF THE POLITICAL DILEMMA AND WHAT COULD BE DONE ABOUT THAT.

Q: I'M TRYING TO LOOK SPECIFICALLY AT WHAT YOU'RE LOOKING FOR IN TERMS OF YOU WOULD LIKE THE PRISONERS OF WAR SETTLED, YOU WOULD LIKE THE GOVERNMENT SETTLED ,- WHAT SPECIFICALLY DO YOU THINK AS A MILITARY MAN WOULD KEEP THE PEACE IN THE FUTURE?

KELLY: FOR THE IRAQI MILITARY, AND IT'S A DIFFERENT PART OF THE PROBLEM, TO AGREE WITH WHAT WE'RE TELLING THEM. INCIDENTALLY, THEY'RE NOT IN MUCH OF A POSITION NOT TO AGREE TO THE TERMS THAT WE'VE LAID OUT THAT THEY HAVE TO ABIDE BY. NOW YOU'VE GOT A NUMBER OF COUNTRIES OVER THERE, AT LEAST TWO, THAT HAVE BEEN PRETTY WELL WRECKED. THERE WILL BE SOME POLITICAL DECISIONS

ASSOCIATED WITH THE EVENTUAL REBUILDING OF THOSE COUNTRIES. THE PRESIDENT SAID TODAY THAT IRAQ IS A WEALTHY COUNTRY, AND IT CAN DEVELOP ITS FUTURE WEALTH INTO PAYING REPARATIONS AND REGENERAT, ING ITS COUNTRY. HE ALSO SAID THAT IN SIMPLE HUMANITARIAN TERMS, IF THEY HAD SEVERE NEEDS, WE WOULD PROBABLY HELP THEM. I'M CERTAIN WE WOULD. IN KUWAIT THE SAME THING IS TRUE. BUT WHAT

/****** BEGINNING OF SECTION 004 ******/
THE SPECIFICS ARE REALLY IS IN NATION,BUILDING, IN REBUILDING THOSE NATIONS, IS NOT A MILITARY DECISION ,- IT'S A POLITI, CAL/DIPLOMATIC DECISION.
Q: RETURNING TO THE EARLIER QUESTION ABOUT SADDAM HUSSEIN, ARE YOU CURRENTLY AWARE OF WHERE HE IS THROUGH YOUR INTELLIGENCE, AND ARE YOU WATCHING HIM SO YOU WOULD BE AWARE IF HE WERE TO MOVE?
KELLY: WE DO NOT KNOW WHERE HE IS -, AT LEAST I DO NOT KNOW WHERE HE IS. WE HAVE NEVER TARGETED SADDAM HUSSEIN. THAT'S THE PLAIN, UNVARNISHED TRUTH. IT'S MY OPINION THAT HE WASN'T ,- MY OPINION ,- THAT HE WASN'T IN ONE OF THOSE BUNKERS, THAT HE WAS IN A CIVILIAN NEIGHBORHOOD, BECAUSE HE KNEW HE WAS SAFE THERE. I'M NOT CERTAIN, THERE MAY HAVE BEEN SOME INDICATIONS THAT HE HAD SOME OF HIS MEETINGS IN CIVILIAN AREAS. WHEN WE FINALLY GET A LOOK INSIDE, AND INCIDENTALLY, YOU'RE GOING TO FIND OUT THAT WE WERE VERY PRECISE IN WHAT WE STRUCK AND WHAT WE DIDN'T STRIKE. BUT I DON'T KNOW WHERE HE IS, AND WE'RE NOT TARGETING HIM.
Q: WHAT ACTION TO TARGET HIM, CAPTURE HIM, OR SIMPLY STOP HIM FROM LEAVING THE COUNTRY ARE YOU PREPARED TO UNDERTAKE?
KELLY: WE HAVE BEEN DIRECTED TO TAKE NO ACTION TO FIND HIM, TO STOP HIM FROM LEAVING THE COUNTRY, OR ANYTHING ELSE. SO WE ARE EXERCISING NORMAL CAUTION. WE GAVE AIRPLANES FLYING OVER BAGHDAD NOT BECAUSE OF SADDAM HUSSEIN, BUT BECAUSE WE WANT TO LET THE PEOPLE THERE KNOW THAT WE HAVE THE CAPABILITY TO REINITIATE COMBAT, SHOULD THAT BECOME NECESSARY. WE ARE NOT ON A HUNT FOR SADDAM HUSSEIN; WE HAVEN'T BEEN DIRECTED TO DO THAT. THE PRESI, DENT SAID HE HAS TO FACE INTERNATIONAL JUSTICE AT SOME POINT, BUT HE HAS NOT GIVEN ANY INSTRUCTIONS TO US TO...
Q: YOU WOULD NOT LET HIM LEAVE, WOULD YOU?
KELLY: IF WE FOUND OUT THAT HE WAS LEAVING, AND WE KNEW ABOUT IT, I WOULD TURN IT OVER TO MY BOSS AND SAY WHAT DO YOU WANT ME TO DO. WE ARE CAPABLE OF DOING ANYTHING YOU WANT.
Q: DO YOU HAVE A COUNT YET ON COALITION VEHICLES LOST -, ARMORED OR OTHERWISE? OR U.S.?
KELLY: NOT COMPLETE. AS I SAID YESTERDAY, I KNOW THE MARINES LOST TWO M-60 TANKS; THE ARMY HAD TWO M1-A1 TANKS DAMAGED. I DON'T HAVE THE TOTALS ON THAT YET, MY GUESS IS IT WILL PROBABLY BE SEVERAL DAYS BEFORE THAT CAME IN.
Q: WHAT KIND OF LEVERAGE DO WE REALLY HAVE AS WE GO TO THESE NEGOTIATIONS? WE SAY WE CAN RESUME FIGHTING, BUT WE'VE DESTROYED 42 OF 42 DIVISIONS. WE SAID WE'RE NOT GOING TO STRIKE CIVILIANS. WHAT CAN WE REALLY DO TO IRAQ THAT WE HAVEN'T ALREADY DONE TO THEM?
KELLY: WE CAN GET THE RESIDUAL OF THE DEFEATED FORCES THAT ARE

IN THE KUWAITI THEATER OF OPERATIONS, AND I DON'T WANT TO GO TOO FAR IN THIS FOR OBVIOUS REASONS, BUT THERE ARE STILL MEANS OF PRODUCTION AND DISTRIBUTION THAT EXIST WITHIN THE COUNTRY THAT COULD BE ATTACKED. IT WOULD DEPEND ON WHAT A NATIONAL DECISION WAS. WE'RE OBVIOUSLY IN A POSITION IN THE MILITARY TO DO JUST ABOUT ANYTHING THAT THE PRESIDENT WANTS TO DO. INCIDENTALLY, WE HAVE SOME CONSIDERABLE LEVERAGE IN THAT WE, RIGHT NOW, POSSESS A CONSIDERABLE AMOUNT OF THAT COUNTRY. WE HAVE FORCES IN THE SOUTHERN THIRD OF THE COUNTRY, AND I WOULD ASSUME THAT THE IRAQIS WOULD WANT THAT PART OF THEIR COUNTRY BACK. THEY'RE GOING TO HAVE TO BE FORTHCOMING IN ORDER TO GET IT BACK.

Q: CAN YOU TALK TO US ABOUT THE REPUBLICAN GUARD FOR A MINUTE? YOU SAID OVER THE FOUR DAYS THERE WERE 29 UNITS, AND IT WORKED ITS WAY DOWN TO ONE UNIT LEFT. WERE ALL THESE GUYS KILLED? DO YOU HAVE ANY IDEA WHAT HAPPENED TO THEM? ALSO, WHAT'S THE SENIOR,MOST OFFICIAL YOU'VE CAPTURED?

KELLY: I THINK A BRIGADIER GENERAL WE SAID YESTERDAY, WHO WAS AN ACTING DIVISION COMMANDER OR A DIVISION COMMANDER. AS WAS INDICATED EARLIER, IT'S POSSIBLE THAT SOME OF THE LEADERSHIP LEFT THEIR UNITS IN THE LURCH WHEN THINGS GOT TOUGH. THE REPUBLICAN GUARDS HAD THREE HEAVY DIVISIONS -, THEY HAVE BEEN DESTROYED. THEY ALSO HAD A NUMBER OF OTHER INFANTRY DIVISIONS WHO HAVE BEEN MANGLED. BUT MANY OF THE INDIVIDUALS FROM THOSE DIVISIONS ARE PROBABLY ON THE ROAD WALKING NORTH ALONG WITH A WHOLE LOT OF OTHER IRAQI SOLDIERS. THEY DO NOT HAVE A FORCE IN THE FIELD THAT'S CAPABLE OF ENGAGING IN COMBAT.

Q: WHEN THEY GET BACK TO IRAQ, DO YOU HAVE ANY BALLPARK ON HOW MANY REPUBLICAN GUARD TROOPS ARE LEFT, ARE ALIVE?

MCCONNELL: IT WOULD BE VERY DIFFICULT TO SAY. THREE HEAVY, FOUR INFANTRY, AND ONE SPECIAL FORCES ,- EIGHT, THAT'S WHAT THEY STARTED WITH. SOME OF THOSE ARE WALKING NORTH. OVER TIME, WE'LL PROBABLY BE ABLE TO MAKE SOME ASSESSMENT ON WHAT'S LEFT. BUT THE POINT I WOULD HIGHLIGHT FOR YOU -, EVEN IF THEY'RE GOING NORTH AND THEY'RE STILL ALIVE, IT'S NOT AN EFFECTIVE FIGHTING FORCE. IT'S NOT SOMETHING WE HAVE TO CONTEND WITH IN THE NEXT FEW MONTHS, OR SIX MONTHS, OR MORE.

KELLY: OR YEARS.

MCCONNELL: THEY WOULD HAVE TO GET SOMEWHERE, GET ORGANIZED, GET WEAPONS, AND SO ON. IF THEY DO ALL THAT, WE'LL KNOW ABOUT IT.

KELLY: AND THEY'D HAVE TO REARM, THEY'D HAVE TO BECOME A TEAM AGAIN, WHICH THEY'RE NOT RIGHT NOW. AS A MATTER OF FACT, IT'S MY STRONGLY-HELD BELIEF THAT WHEN THIS DEFEATED ARMY GETS BACK TO BAGHDAD, THEY'RE GOING TO BE PRETTY MAD.

Q: THERE WAS SOME DISCUSSION AS THE END NEARED, THAT THE SOVIETS MIGHT HAVE BEEN TRYING TO FRUSTRATE A COMPLETE DEFEAT OF SADDAM HUSSEIN THROUGH DIPLOMATIC INITIATIVES. ON THE OTHER HAND, IT WOULD APPEAR THAT THEY DID NOT GIVE THE IRAQIS WHAT WOULD APPEAR TO BE VERY GOOD SATELLITE COVERAGE OF THE VII CORPS' FLANKING MANEUVER, AND OUR POSITIONS ON THE FIELD. DO YOU ASSUME THAT, IN FACT, IS WHAT DID HAPPEN, THAT THEY DIDN'T TELL THEM?

KELLY: I SIMPLY DON'T KNOW. I DON'T KNOW WHETHER THEY TOLD THEM

QR NOT. IT CERTAINLY APPEARS THAT THEY DIDN'T. AS MIKE POINTED OUT YESTERDAY, EVEN HAD THEY KNOWN, THEIR ABILITY AT THAT POINT TO SHIFT FORCES AND COUNTER IT WAS QUITE LIMITED. BUT THEY DID NOTHING TO SHIFT FORCES. SO IT APPEARS THEY DIDN'T KNOW. IF YOU DERIVE FROM THAT THAT THAT'S BECAUSE THE SOVIETS DIDN'T TELL THEM, SO BE IT. I DON'T KNOW THAT TO BE A FACT.
Q: IS THERE ANY INTELLIGENCE AVAILABLE NOW ABOUT WHY THE IRAQI AIR FORCE MIGRATED TO IRAN? IS THERE ANY SPECIFIC REASON? AND WHAT HAPPENS IF THEY START TO MIGRATE BACK?
KELLY: THE ANSWER TO THE FIRST PART IS, I DON'T KNOW THAT WE KNOW ANYTHING FURTHER THAN WE KNEW BEFORE.
Q: NO CLUE AT ALL AS TO WHY THEY DID THAT?
KELLY: BECAUSE WE DON'T HAVE A PRESENCE IN IRAN, AND WE'RE NOT ABLE TO QUESTION THE PEOPLE. IT'S MY BELIEF THAT SOME OF THEN BUGGED OUT, CLEARLY. IT'S ALSO MY BELIEF NOW THAT IRAQ IS GOING TO HAVE TO NEGOTIATE WITH IRAN TO GET THOSE AIRPLANES BACK. I THINK I ALLUDED TO THAT BEFORE, AND I DON'T KNOW THAT THEY'RE GOING TO BE SUCCESSFUL.

/****** BEGINNING OF SECTION 005 ******/
MR. WILLIAMS: THANK YOU VERY MUCH.
I DON'T KNOW YET WHAT OUR PLAN, BY THE WAY, IS FOR BRIEFINGS NEXT WEEK. WE WILL NOT BRIEF HERE IN THE PENTAGON TOMORROW, AND I DON'T KNOW YET WHETHER THERE WILL BE ANY STATEMENT AFTER THE MEETING IN THE DESERT TOMORROW ,- WHETHER GENERAL SCHWARZKOPF OR PRINCE KHALID OR ANY OF THE OTHER COALITION REPRESENTATIVES WILL HAVE ANY KIND OF STATEMENT AFTER THE MEETING.
JUST A COUPLE OF QUICK THINGS HERE. IT'S BEEN AWHILE SINCE WE'VE GOTTEN A REQUEST TO GET THE RESERVIST NUMBERS. THE NUMBER OF GUARD AND RESERVISTS ON ACTIVE DUTY TO DATE -, TOTAL NUMBER IS 225,258. IF YOU GO DOWN BY SERVICE: FOR ARMY, 142,968; FOR NAVY, 18,930; FOR AIR FORCE, 33,566; FOR THE MARINE CORPS, 28,917; AND FOR THE COAST GUARD, 877. THE STATISTIC THAT WE'VE HAD ALL ALONG HERE, THAT ABOUT 15 PERCENT OF THE SERVICE MEMBERS WHO ARE IN DESERT STORM IN THE FIELD OF OPERATIONS ARE MEMBERS OF THE GUARD AND RESERVE. OF COURSE, ALL MEMBERS OF THE GUARD AND RESERVE IN THIS NUMBER HAVE BEEN CALLED TO ACTIVE DUTY TO SUPPORT THIS OPERATION. THEY HAVEN'T ALL NECESSARILY GONE TO THE PERSIAN GULF -, SOME OF THEM ARE BACKFILLS FOR UNITS THAT WENT OVER THERE, BUT THEY'VE ALL BEEN CALLED TO DUTY SPECIFICALLY FOR THAT OPERATION.
Q: YOU SAID IN THE COVERAGE OF THE IRAQI AND ALLIED MEETING TOMORROW IN SOUTHERN IRAQ THAT ALL INTERNATIONAL MEDIA WOULD BE REPRESENTED. ARE YOU REFERRING TO THE FACT THAT THERE WILL BE ONLY POOLS THERE, OR WILL THERE BE UNILATERAL COVERAGE?
A: NO, I DON'T THINK THERE WILL BE MUCH, WELL, IT WILL BE SORT OF A MODIFIED WHITE HOUSE POOL TYPE DEAL. THERE'S CLEARLY NO WAY, THIS MEETING IS HAPPENING AT A SPOT THAT IS, FOR ALL INTENTS AND PURPOSES, A BATTLEFIELD STILL. IT'S HAPPENING SORT OF OUT IN THE MIDDLE OF NOWHERE. THERE ARE STILL ACTIVE MINEFIELDS IN THE AREA. I DON'T KNOW IF THERE ARE ANY AIR BASES NEARBY THAT CAN BE

USED WITH ACTIVE RUNWAYS. SO YOU JUST CAN'T HAVE A COUPLE OF HUNDRED FOLKS THERE, AS YOU'D LIKE TO HAVE. WE'LL TRY TO MAKE ARRANGEMENTS FOR EVERYBODY TO BE REPRESENTED, BUT I DON'T KNOW EXACTLY HOW THAT'S GOING TO WORK.

Q: CAN YOU DESCRIBE GENERALLY A SEQUENCE OF EVENTS OF WHAT IS BEING PLANNED, WHAT WILL HAPPEN AND WHEN, WHAT THE SCENARIO AND THE ENVIRONMENT LOOK LIKE?

A: I CAN'T GIVE YOU MUCH DETAIL. YOU'LL BE ABLE TO SEE, I SUSPECT, SOME KIND OF ARRIVAL. THEY'LL WALK INTO WHEREVER THEY MEET -, WHETHER IT'S A BUILDING OR A TENT OR WHATEVER IT IS. THERE WILL BE A PHOTO OPPORTUNITY WHEN THEY'RE SEATED AT THE TABLE. OBVIOUSLY, THAT WILL BE A SMALL GROUP THAT YOU CAN TAKE INSIDE WHEREVER THEY MEET. THEN THE SMALL PHOTO POOL WILL LEAVE, THEN THE MEETING WILL GO ON, AND AT SOME POINT IT WILL END. I DON'T KNOW IF IT'S GOING TO BE OVER A COUPLE OF DAYS OR WHAT, BUT WHEN IT ENDS THAT DAY, THEN YOU'LL BE ABLE TO SEE THEM LEAVE. AGAIN, I DON'T KNOW WHETHER THERE WILL BE A STATEMENT OR NOT. I CAN'T REALLY GIVE YOU MANY DETAILS, OTHER THAN THAT.

Q: DO YOU KNOW WHO'S GOING TO BE THERE FOR THE ALLIES?

A: THE PRESIDENT SAID IT WILL BE GENERAL SCHWARZKOPF AND PRINCE KHALID, WHO IS THE COMMANDER OF THE SAUDI FORCES. I THINK THE BRITISH SAID THAT GENERAL DELABILLIERE, HOWEVER YOU PRONOUNCE HIS NAME, WILL BE THERE. BEYOND THAT, I'M NOT CERTAIN WHO'S GOING TO BE THERE.

Q: CAN YOU ADDRESS THE NUMBER OF RESERVE GROUPS BEING ACTIVATED? THERE WERE REPORTS THIS MORNING ABOUT A WHOLE LOAD OF RESERVISTS BEING ACTIVATED AT THIS POINT.

A: WE WILL FOLLOW OUR CONTINUING POLICY THAT WE HAVE SINCE THE BEGINNING OF OPERATION DESERT SHIELD, AND SINCE THE FIRST GUARDS, MEN AND RESERVISTS WERE CALLED UP AT THE END OF AUGUST, WHICH IS WE WILL ANNOUNCE ANY CALLUPS AS THEY GO. SO IF WE HAVEN'T ANNOUNCED IT, THEY HAVEN'T BEEN CALLED UP.

Q: ARE YOU EXPECTING MORE...

A: NOT THAT I KNOW OF, BUT THERE COULD BE SOME.

Q: GIVEN OUR SUPERIORITY AND DOMINANCE OF THE AIR, HOW ARE THESE IRAQI REPRESENTATIVES GOING TO GET THERE? WOULD THEY HAVE TO COME BY LAND, OR WILL SOME ARRANGEMENTS BE MADE TO FLY THEM IN?

A: MY GUESS IS IT WOULD BE BY LAND. I'M NOT POSITIVE ABOUT THAT. THAT'S STILL TRYING TO BE WORKED OUT.

Q: CAN YOU FIND OUT THE EXACT NUMBER OF MEDICAL RESERVISTS CALLED UP FOR OPERATION DESERT STORM? SECONDLY, I'M SURE YOU'VE BEEN SEEING ALL THESE REPORTS ABOUT THE DOCTORS, AS FAR AS OPERA, TION DESERT STORM, MANY SAYING THAT AFTER THE WAR THEY PLAN TO POSSIBLY GET OUT OF THE RESERVES BECAUSE THERE'S NO INCENTIVE BECAUSE THE PAY IS NOT COMPARABLE TO PRIVATE PRACTICE. CAN YOU SEE ANY TYPE OF LEGISLATION OR SOMETHING THAT WILL GIVE A STRON, GER MEDICAL ARM TO THE MILITARY IF THIS DOES HAPPEN?

A: NUMBER ONE, NO, I CAN'T GIVE YOU AN EXACT NUMBER OF MEDICAL PERSONNEL. WE COULD TRY TO DO THAT ,- IT'S GOING TO TAKE AWHILE. WE DON'T REALLY KEEP TRACK OF THE NUMBER OF RESERVES BY SPECIAL- IZATION. YOU'VE GOT MEDICAL PERSONNEL, YOU'VE GOT LINGUISTS,

YOU'VE GOT WATER PURIFICATION SPECIALISTS, LOGISTICS -, TRYING TO BREAK ALL THAT OUT WOULD BE A PRETTY MASSIVE UNDERTAKING. I CAN'T GUESS WHAT SORT OF LEGISLATION MIGHT BE INTRODUCED IN THE CONGRESS, EITHER THE HOUSE OR THE SENATE. CLEARLY, THERE WERE RESERVISTS WHO WERE ON RESERVE THAT NEVER THOUGHT THEY'D GET CALLED TO ACTIVE DUTY. LIKE ONE OF YOU SAID EARLIER, IT'S LIKE SOMEBODY JOINING THE POSTAL SERVICE, AND THEN BEING SHOCKED THAT THEY HAVE TO DELIVER SOME MAIL. SO THERE WAS A LITTLE ATTITUDE READJUSTMENT, I KNOW. IT'S GOING TO BE AN IMPORTANT THING. THAT'S CLEARLY GOING TO BE ONE OF THE THINGS WE'LL HAVE TO LOOK AT WHEN THIS IS ALL OVER. IT'S VITAL TO THE FORCE THAT WE HAVE AN ADEQUATE MEDICAL SUPPORT STAFF IN RESERVE. HOW WE DO IT, I'M SURE IS SOMETHING THAT PEOPLE ARE GOING TO BE LOOKING AT. BUT I CAN'T GUESS NOW HOW IT WOULD BE.

Q: DOES THE DEPARTMENT OF DEFENSE NOW CONSIDER THAT THE GULF WAR IS OVER? IF NOT, AT WHAT POINT ARE YOU ABLE TO OFFICIALLY SAY THE WAR HAS ENDED?

A: IT AIN'T OVER 'TIL THE GUY IN THE WHITE HOUSE SINGS, BASICALLY. (LAUGHTER) AND THE OTHER MEMBERS OF THE COALITION. RIGHT NOW WHAT WE HAVE UNDER THE TERMS THAT THE PRESIDENT HAS SPELLED OUT IS A TEMPORARY STOP, OR A SUSPENSION OF OFFENSIVE ACTIVITIES SUBJECT TO THE IRAQIS FIRST OF ALL, AGREEING TO THIS MEETING; AND SECONDLY, AGREEING TO THE TERMS THAT THE PRESIDENT HAS SPELLED OUT. UNTIL THAT HAPPENS, THE ORDERS WE HAVE RIGHT NOW, WHICH WE, IN TURN, HAVE FORWARDED TO THE COMMANDERS IN THE FIELD, SAY THAT RIGHT NOW WE HAVE A SUSPENSION IN OFFENSIVE OPERATIONS. UNTIL THE PRESIDENT OF THE UNITED STATES ORDERS US OTHERWISE, THAT'S THE POSTURE THAT WE'RE IN RIGHT NOW.

Q: DO YOU HAVE ANY MORE DETAILS ON THE TWO DOCTORS WHO DIED AFTER HITTING THE MINEFIELD?

/****** BEGINNING OF SECTION 006 ******/

A: I DON'T. I UNDERSTAND THAT IT HAPPENED, THEY WERE IN A JEEP OR A HUMVEE. THEY HIT A MINE, AND THEN SOMEBODY GOT OUT OF THE JEEP, AND THAT PERSON STEPPED ON A MINE. BUT OTHER THAN THAT, I DON'T KNOW ANY DETAILS BEYOND THAT. I DON'T KNOW WHERE IT WAS OR ANYTHING LIKE THAT.

Q: CAN YOU TAKE THE QUESTION?

A: OBVIOUSLY, WHEN WE GET MORE INFORMATION WE'LL PUT IT OUT.

Q: THE PRESIDENT SAID WE'RE GOING TO MEET TOMORROW AFTERNOON. I ASSUME HE MEANT TOMORROW AFTERNOON SAUDI ARABIAN TIME?

A: I WOULD ASSUME SO. IF HE MEANT TOMORROW AFTERNOON EASTERN TIME, THAT WOULD BE IN THE MIDDLE OF THE NIGHT IN SAUDI ARABIA. SO I ASSUME THAT IT'S TOMORROW AFTERNOON PERSIAN GULF TIME.

Q: YOU DON'T HAVE ANY IDEA OF EARLY AFTERNOON, LATE AFTERNOON?

A: I DON'T.

Q: DO WE KNOW HOW MANY OF THE POW'S OR MIA'S ARE WOMEN?

A: I THINK I KNOW OF ONE WOMAN THAT'S MISSING. THAT WAS THE TWO PERSON CREW THAT WAS DRIVING ALONG THE TAPLINE ROAD IN A VEHICLE, AND THAT WAS THE LAST TIME THEY WERE SEEN. THAT'S A MEMBER OF THE ARMY. BUT I DON'T THINK THERE ARE ANY MORE BEYOND THAT. ARE

ABORTED MILITARY EFFORT AT KHAFJI HAPPENED. THE WHOLE FORCE LEFT TO GO DOWN AND CARRY OUT THE KHAFJI OPERATION, AND THEY DIDN'T ALL SHOW BACK UP AGAIN.
Q: IT WOULD APPEAR THE MILITARY, IF ONLY TO DETERMINE FOR ITS OWN PURPOSES THE EFFECTIVENESS OF SUCH MASSIVE, NON,STOP BOMBING, WOULD WANT SOMETHING, AND ALSO WOULD WANT SOMETHING IN CASE THE OTHER SIDE -, IF SADDAM HUSSEIN SURVIVES ,, GIVES SOME OUTRAGEOUS FIGURE FOR DEAD TO SUIT HIS PURPOSES.
A: IT MAY WELL HAPPEN. I CAN'T CONCEIVE OF A MILITARY OPERATION OF THIS MAGNITUDE IN WHICH WE'D HAVE ANY NUMBER THAT WE WOULD BE CONFIDENT OF WITHIN 48 HOURS OF WHEN HOSTILITIES STOPPED.
Q: CAN YOU PLEASE DESCRIBE WHAT TYPE OF RESIDUAL FORCES THE U.S. WOULD LIKE TO HAVE IN PLACE IN THE REGION, AND WHAT THE FUTURE MAY LOOK LIKE AS FAR AS THE U.S. MILITARY IS CONCERNED?
A: LET'S TALK SHORT TERM AND LONG TERM. SHORT TERM, IN TERMS OF BRINGING THE BOYS HOME -, AND WOMEN, OF COURSE. I'M USING THAT TERM METAPHORICALLY. THERE'S LOTS OF WORK THAT NEEDS TO BE DONE. THERE IS, FIRST OF ALL, THE FACT THAT AS I DISCUSSED WITH KEENAN A MOMENT AGO, ALL WE HAVE IS A SUSPENSION OF HOSTILITIES RIGHT NOW ,, WEE HAVE A SUSPENSION OF OFFENSIVE ACTIVITIES. AT SOME POINT, LET'S ASSUME THEN WE FORMALLY END THE CONFLICT AND THERE IS SOME KIND OF DOCUMENT SIGNED THAT BRINGS IT TO AN END. THEN YOU'VE GOT TO HAVE SOME CLEARING OF THE BATTLEFIELD -, MINES, BOOBY TRAPS, EXPLOSIVES AND SO FORTH, IN ORDER FOR UNITS TO MOVE AROUND SAFELY AND DEPART SAFELY. THE UNITS WILL HAVE TO LEAVE THE BATTLEFIELD IN SOME KIND OF ORDERLY SEQUENCE. IN OTHER WORDS, YOU'VE GOT TO HAVE A COVERING FORCE THAT STAYS IN PLACE TO PROTECT DEPARTING TROOPS ,- THAT'S JUST THE STANDARD WAY OF DOING BUSINESS. YOU HAVE TO HAVE SOME KIND OF ORDERLY FLOW BACK TO THE PORTS -- WHETHER IT BE AN AIR BASE OR A NAVAL PORT. IT JUST TAKES LONGER TO LOAD THAT STUFF UP THAN IT DOES TO UNLOAD IT. IT'S KIND OF LIKE PACKING YOUR CAR. IT TAKES LONGER TO PACK IT THAN IT DOES TO UNPACK IT. YOU'VE GOT TO PREPARE THE EQUIPMENT FOR SHIPMENT. VEHICLES HAVE TO BE WASHED, EVERYTHING HAS TO BE TIED DOWN, ALL THAT STUFF HAS TO BE SECURED. YOU'LL REMEMBER THE EXTENSIVE PREPARATIONS THAT WENT INTO PUTTING THAT STUFF ON THE SHIPS, YOU HAVE TO BALANCE THE LOAD AND HAVE THE HEAVY STUFF IN THE MIDDLE AND ALL THAT KIND OF THING. YOU HAVE TO SHIP THE AMMUNITION BACK, STORE IT, IT HAS TO BE STORED SAFELY TO GO BACK, AND ALL THAT SORT OF STUFF. SO THAT HAS TO ALL TAKE PLACE. THAT'S THE SHORT TERM.
THEN IN TERMS OF THE LONG TERM, AND WHAT KIND OF LONG TERM FORCE

/****** BEGINNING OF SECTION 007 ******/
YOU'RE GOING TO HAVE THERE, LET ME GIVE YOU THE SORT OF GENERAL CONCEPT. NUMBER ONE, YOU'LL PROBABLY HAVE, AS WE'VE SAID BEFORE HERE, A HIGHER THAN NORMAL NAVAL PRESENCE -, AT LEAST HIGHER THAN THERE WAS BEFORE THE CONFLICT, MOST LIKELY. PERHAPS SOME KIND OF AIR PRESENCE THERE; PERHAPS, IN THE SHORT TERM, SOME KIND OF GROUND PRESENCE. IT COULD BE A MULTINATIONAL FORCE TO KEEP THE SECURITY OF KUWAIT UNDER WATCH. BUT BEYOND THAT, THAT'S KIND OF

THE INTERIM. THE LONG TERM, WHICH IS THE ONE YOU ASKED, I THINK THE ANSWER TO THAT WON'T BE KNOWN UNTIL SECRETARY BAKER BEGINS HIS TRIP. HE'S GOING TO START TO TALK ABOUT SOME OF THOSE SUB, JECTS WITH THE COUNTRIES IN THE REGION. WHAT SORT OF LONG TERM SECURITY ROLE WILL WE HAVE THERE. THAT'S ONE OF THE THINGS ON HIS AGENDA, AND I DON'T KNOW WHAT THE ANSWER IS TO THAT.

Q: GENERAL KELLY ALLUDED TO THIS. WHAT HAPPENS IF THE MEETING TOMORROW BREAKS OFF, IF THE IRAQIS REFUSE TO NEGOTIATE, THE MEETING BREAKS OFF? ALL OF THOSE FORCES IN THE FIELD THAT HAVE SORT OF BEEN MENTALLY PACKING THEIR BAGS READY TO GO HOME, DOES THAT MEAN THAT IF THAT MEETING BREAKS OFF THEY SHOULD NOT COUNT ON GETTING HOME ANY TIME SOON?

A: THERE'S A LOT OF INTEREST HERE IN GETTING EVERYBODY HOME, BECAUSE FAMILIES MISS THOSE FOLKS, AND WE'RE ALL LOOKING FORWARD TO GETTING THEM HOME, AND WELCOMING THEM HOME. BUT I'D BE SUR PRISED IF ALL THE TROOPS OUT THERE ARE EAGER TO LEAVE. I THINK THEY WANT TO MAKE SURE THAT THIS, THEY DON'T WANT TO GET THIS CLOSE AND HAVE SOMETHING HAPPEN, AND HAVE IT ALL UNRAVEL. SO I DON'T THINK THE FORCES ARE URGENTLY LOOKING TO LEAVE UNTIL THEY'RE SURE THAT THE JOB IS DONE.

Q: AGAIN, IF THE MEETING BREAKS OFF AND IT'S NOT ACCORDING TO THE WAY YOU WOULD HOPE IT MIGHT BE, THIS MEANS THAT BASICALLY THEY'RE GOING TO BE OUT THERE UNTIL YOU DO GET A MEETING THAT IS SUCCESSFUL.

A: THAT'S GOING TO BE SOMETHING THAT THE PRESIDENT WILL TELL US. OUR ORDERS COME FROM THE COMMANDER-IN-CHIEF. RIGHT NOW THEY ARE SUSPEND HOSTILITIES, RELOAD, REARM, BE VIGILANT, MAINTAIN THE RECONNAISSANCE, KEEP AN EYE ON THINGS, AND THAT'S THE POSTURE WE'RE IN RIGHT NOW. BUT WHAT HAPPENS IF THINGS GO SOUR THERE, I DON'T KNOW WHAT THE ANSWER IS.

Q: ARE YOU CONCERNED, OR HAS THE SECRETARY EXPRESSED ANY CONCERN OVER THE PAST DAY OR SO THAT THERE MIGHT BE, THAT THE PRESSURE FROM THE PRESS AND MAYBE THE PUBLIC, OR THE PERCEIVED PRESSURE, TO GET THE TROOPS HOME? YOU HEAR IT OVER AND OVER AGAIN, IT MIGHT BE A LITTLE PREMATURE, TO SAY THE LEAST. ARE YOU A LITTLE CONCERNED ABOUT THE MILITARY OPERATION AND, DO YOU THINK WE'RE BEING A LITTLE HEAVY-HANDED, I GUESS IS WHAT I'M ASKING YOU?

A: NO, NOT AT ALL. I THINK IT'S SOMETHING THAT WE ALL UNDER, STAND. PEOPLE HERE IN THE DEFENSE DEPARTMENT HAVE FAMILIES, AND THERE ARE A LOT OF PEOPLE THAT I TALK TO EVERY DAY IN THIS BUILD, ING WHO HAVE SONS AND DAUGHTERS OVER IN THE PERSIAN GULF WHO UNDERSTAND THIS EVERY BIT AS WELL AS EVERYBODY ELSE. WE THINK IT'S GOOD THAT FOLKS IN THE UNITED STATES ARE SAYING LET'S GET THEM HOME, WE WANT TO WELCOME THEM, THEY'VE DONE WELL, THAT'S A GREAT POSITION FOR US TO BE. BUT PERHAPS BECAUSE OF THE FACT THAT THE GROUND WAR LASTED FOUR DAYS, THERE'S NOW KIND OF INSTANT CAMERA IDEA OF HOW THIS WORKS. IT'S NOT OVER YET. WE HAVE A SUSPENSION OF HOSTILITIES. WE WANT TO MAKE SURE THAT IT ENDS PROPERLY AND THAT THE NECESSARY THINGS ARE DONE TO GET THE TROOPS HOME SAFELY, GET THE EQUIPMENT BACK WHERE IT BELONGS, AND HAVE A PEADY FORCE WHEN WE GET BACK. THAT'S GOING TO TAKE SOME TIME.

WE KNOW HOW EAGER THE FAMILIES ARE, BUT AS I SAID A MOMENT AGO, IT'S NOT EVEN BEEN 48 HOURS SINCE THE THING WENT INTO A SUSPENSION OF HOSTILITIES, AND NOW WE'RE TALKING ABOUT GETTING THEM ALL HOME. SO IT'S GOING TO TAKE AWHILE, BUT I DON'T THINK ANYBODY HERE FEELS UNDER EXTRAORDINARY PRESSURE TO DO THAT.

Q: CAN YOU ADDRESS THE REPORTS LAST NIGHT THAT CAME OUT... THE PENTAGON OFFICIALLY SAID WE DON'T HAVE A PLAN ON BRINGING HOME TROOPS AND RESERVES, AND WE'RE WORKING ON A PLAN. THERE WERE SPECIFIC UNITS REPORTED IN THE PRESS LAST NIGHT ABOUT WHICH ONES WERE GOING TO BE HOME SOON, THE FASTEST. CAN YOU TALK TO US ABOUT WHAT EXACTLY WE KNOW AT THIS POINT, AND WHAT THAT PLAN IS, AND WHEN IT WILL BE FINALIZED?

A: I'M HAPPY TO EXPLAIN THAT. YOU DON'T HAVE A WASHINGTON POST UNTIL THE PRESSES START TO ROLL. THE STORY ISN'T READY TO GO UNTIL YOUR EDITOR SIGNS OFF ON IT. IT WORKS THE SAME WAY HERE. THE MILITARY DEPARTMENTS COME UP WITH A PLAN, THEY SUBMIT THE PLAN TO THE LEADERSHIP, AND IT DOESN'T GO INTO EFFECT UNTIL THE SENIOR LEADERSHIP OF THE BUILDING ,, GENERAL POWELL AND SECRETARY CHENEY ,, APPROVE IT. UNTIL THEY DO, THERE IS NO PLAN. NOW I SUSPECT THAT WHAT'S HAPPENING IS THAT THE FOLKS IN THIS BUILDING WHO KNOW HOW TO REPORT HERE, GO OUT AND TALK TO THE CAPTAINS AND THE LIEUTENANTS AND THE SERGEANTS WHO HAVE TO DRAFT THIS PLAN, AND THEY SAY HEY, WHAT ARE YOU WORKING ON THERE, SERGEANT BILCO? AND HE SHARES WITH THEM HIS IDEAS FOR HOW TO END THE WAR. SOME OF THAT STUFF MAY BE ADOPTED, AND SOME OF IT MAY NOT BE. BUT THERE IS NO PLAN UNTIL SECRETARY CHENEY AND GENERAL POWELL APPROVE IT, AND THEY HAVEN'T DONE SO YET. BUT IT'S OBVIOUS SOMETHING THAT THEY'RE WORKING ON.

Q: GENERAL KELLY TALKED ABOUT THESE CONVOYS HEADED BACK TO BAGHDAD, THE POSSIBLE EFFECTS OF WHAT WILL HAPPEN WHEN THEY GET THERE, WHAT HAPPENED WHEN THEY FACED THE IMPERIAL FORCES. IS THERE ANY PLAN FOR THE UNITED STATES OR THE COALITION TO STEP IN IF THERE'S A VACUUM, AN IMPLOSION OF LEADERSHIP, OR ANY INSTABIL, ITY IN BAGHDAD OF THE IRAQI GOVERNMENT? SOME SORT OF LAW AND ORDER ROLE THE COALITION MIGHT PLAY.

A: NOT THAT I KNOW OF, NO.

Q: SECRETARY CHENEY SAID ABOUT FOUR WEEKS AGO DURING THE BUDGET TALKS THAT THE BATTLESHIPS WILL BE DECOMMISSIONED. IN LIGHT OF THE PERFORMANCE OF THE MISSOURI AND THE WISCONSIN AND THE OTHER SHIPS HAVE DONE, IS THERE ANY RETHINKING ABOUT DECOMMISSIONING THE BATTLESHIPS, IN LIGHT THAT THEY DID SUCH A GOOD JOB IN THE PERSIAN GULF?

A: NO, I DON'T THINK SO. THERE WAS NO DOUBT IN OUR MINDS THAT THE BATTLESHIPS WOULD DO A GOOD JOB. THERE'S NO DOUBT IN OUR MINDS THAT THE CURRENT SIZE OF THE MILITARY FORCE WOULD DO A GOOD JOB. BUT WE HAVE A PROBLEM, AND THAT IS THAT WE HAVE TO CUT THE BUDGET. CONGRESS' BUDGET THAT THEY'RE GIVING FOR DEFENSE IS GOING DOWN OVER THE NEXT SEVERAL YEARS, SO YOU HAVE TO DECIDED, YOU HAVE TO MAKE PRIORITIES. BATTLESHIPS ARE VERY LABOR INTEN, SIVE. THEY'RE WONDERFUL OLD SHIPS. THEY HAVE BEAUTIFUL, THICK HULLS THAT MAKE THEM EXTREMELY DIFFICULT TO SINK. THEY'VE DONE

AN EX RAORDINARILY GOOD JOB ,- THE MISSOURI AND WISCONSIN BOTH THAT ARE ON STATION THERE ARE EXCELLENT SHIPS. BUT WE JUST CAN'T AFFORD TO KEEP THEM. SO I DOUBT THERE WOULD BE ANY CHANGES.

/****** BEGINNING OF SECTION 008 ******/
Q: WHY NOT SELL THEM? THE KUWAITIS HAVE ALL THIS MONEY...
(LAUGHTER)
A: I DON'T KNOW WHAT WILL HAPPEN TO THEM. I DON'T KNOW HOW WE UNLOAD A BATTLESHIP. IT'S NOT SOMETHING WE DO VERY OFTEN.
PRESS: THANK YOU.
ADMIN
BT

#8898

NNNN

TOPICAL BIBLIOGRAPHY OF PUBLISHED WORKS REGARDING THE HEALTH OF VETERANS OF THE PERSIAN GULF WAR

C. M. Cornelison

G. C. Gray

Technical Document
95-3C

NAVAL HEALTH RESEARCH CENTER
P.O. BOX 85122
SAN DIEGO, CALIFORNIA 92186-5122

NAVAL MEDICAL RESEARCH AND DEVELOPMENT COMMAND
BETHESDA, MARYLAND

#567

Topical Bibliography of Published Works Regarding the Health of Veterans of the Persian Gulf War

Colleen M. Cornelison

CDR Gregory C. Gray, MC, USN

Emerging Illness Research Team
Division of Clinical Epidemiology
Naval Health Research Center
P.O. Box 85122
San Diego, CA 92186-5122

Technical Document No. 95-3C was supported by the Department of Defense/ Health Affairs and funded under the Naval Medical Research and Development Command work unit number 63706N P4464.001-6423. The views presented are those of the authors and do not reflect the official policy of the Department of the Navy, Department of Defense, nor the U.S. Government. Approval for public release; distribution unlimited.

Table of Contents

6

Clinical Considerations in Nerve Agent Intoxication[1]

Frederick R. Sidell

[1]The views expressed in this chapter are those of the author and should not be construed as the position or policy of the Department of the Army unless so designated by other authorized documents.

I. Summary

The history of nerve agents is reviewed in this chapter and a brief summary of their biological activity is provided (a more complete discussion was in a previous chapter). The signs and symptoms caused by exposure to differing amounts of these agents by vapor or by skin contact with liquid are described. Current therapy is detailed, including the use of pretreatment and an anticonvulsant. Finally, recommendations for patient management, based on the author's experience, are given.

II. Introduction

Nerve agents were developed over five decades ago for military use and continue to be a significant threat on the battlefields of the world, or as terrorist weapons. First developed in secrecy before World War II, these agents are now in the armamentariums of several major world powers and those of several smaller nations. In particular, Iraq had a manufacturing capability and used these agents against Iranian military forces and against Iraqi Kurds, in at least one attack on a civilian population. Nerve agents were felt to be a major threat during the war with Iraq.

Much of the clinical information on these agents is from research in the decades after World War II and from published accounts of accidental exposures. A previous chapter discussed the mechanism of action and basic pharmacology of these agents, and this chapter will address the clinical aspects.

III. History and Military Significance

Nerve agents are the most potent of a group of compounds that cause biological effects by inhibiting the enzyme acetylcholinesterase (AChE). The two largest chemical classes of these compounds are the carbamates and the derivatives of phosphorus acids, or organophosphorus (OP) compounds. Probably the first such substance used was the Calabar bean as an *ordeal poison* by the natives of the Calabar region of western Africa (Koelle, 1975; Davis, 1985). The extract of this, used medicinally (Fraser, 1863), was isolated in 1864 by Jobst and Hesse and called physostigmine and isolated again independently in 1865 by Lee and Levin, who called it eserine (Koelle, 1975). This carbamate is still in medicinal use.

The first OP cholinesterase inhibitor was probably tetraethyl pyrophosphate (TEPP), synthesized by Wurtz and tasted (with no ill effects) by Clermont in 1854 (Holmstedt, 1963). Although there were numerous im-

portant advances in OP chemistry over the following decades, the extreme potency of these compounds apparently was first reported in 1932 by Lange and Krueger (who noted the effects of the vapors on themselves) (Koelle, 1975; Holmstedt, 1963).

In the early 1930s the German firm IG Farbenindustrie developed an interest in this group of compounds as possible pesticides and named Dr. Gerhard Schrader to head the research and development effort. In December 1936 Schrader synthesized tabun, or GA (Harris and Paxman, 1982; Robinson, 1971), and noted in himself the effects of its vapors (miosis). A year or so later, Schrader synthesized sarin, or GB (allegedly named in honor of the people instrumental in its development and production, *S*chrader, *A*mbros, *R*udriger, and van der L*in*de (Harris and Paxman, 1982).

Their extreme toxicity made these compounds ideal for use as warfare agents, and in 1940, a production facility was begun at Dyhernfurth. Tabun was first mass-produced in 1942 (Harris and Paxman, 1982; Robinson, 1971), and sarin was later produced there and possibly at another facility at Falkenhagen (Robinson, 1971).

Although the estimates vary, about 10,000 to 30,000 tons of tabun and smaller quantities of sarin (5–10 tons) were produced and put into munitions by the Germans (Robinson, 1971). Why they were never used remains a matter of conjecture, as the Allies did not know of these agents and had no protection or antidotes for them. It is possible their use could have changed the outcome of that war. Among the suggested reasons are that Hitler had been the victim of a chemical agent (mustard) in World War I and found chemical use distasteful; the senior German officers had been in World War I and also disliked them; and the Germans thought that the Allies had these agents and would retaliate with them. A more realistic reason is that in the latter stages of the war, including at the time of the Allied invasion of Europe, Germany lacked the air superiority needed for large-scale delivery.

In the waning stages of the war, the Russians captured the Dyhernfurth facility and moved it, along with key personnel, to Russia where production of the agents resumed in 1946 (Robinson, 1971; Koelle, 1981). At Raubkammer (a German testing facility), United States and British forces captured munitions containing a liquid unknown to scientists in those countries. Over a single weekend, British scientists, working with miosis from accidental exposure to the vapor, elucidated the pharmacology and toxicity of this material (tabun; GA) and documented the anti-dotal activity of atropine (K. M. Wilson; personal communication, 1970).

Records captured later indicated that the Germans had tested nerve agents on inmates of concentration camps to investigate their effects and toxicity and to test antidotes (Wills and DeArmon, 1954). There were casualties, fatal and nonfatal, at Dyhernfurth, and the medical staff there had also developed antidotes (Harris and Paxman, 1982).

A third agent, soman (GD) was synthesized by Dr. Richard Kuhn in 1944 in Germany (Robinson, 1971). Again this discovery was serendipitous in a search for a better insecticide. In a similar search, VX was first synthesized by a British chemical concern in 1954 (Robinson, 1971).

The United States produced sarin from the early 1950s until the early 1960s (Robinson, 1971) and produced VX from 1961 to 1968 (Robinson, 1971). The inventory today consists of these two agents in 23- to 40-year-old rockets, land mines, projectiles, bombs, and bulk containers (Anonymous, 1988). These are stored at six depots within the United States and one depot and demilitarization facility outside the continental United States (Smith, 1989), where destruction of these weapons has been underway since early 1990.

The exact types and amounts of chemical agents stockpiled by other countries are unknown. Russia is known to have a large stockpile, and possibly these were shared with members of the former Warsaw Pact. France has been reported to have chemical weapons. Many Third World countries undoubtedly possess a smaller stockpile, and Libya (at Rabta) and Iraq (at Samarra) have or had large manufacturing facilities.

IV. Toxicity

The four major nerve agents are tabun (GA), sarin (GB), soman (GD), and VX. Their structures and toxicological data in animals were presented in the previous chapter. Estimates of their toxicity in humans vary from source to source, but in general the LCt_{50}'s are estimated to be 400 mg·min/m^3 for tabun, 100 mg·min/m^3 for sarin, 50 mg·min/m^3 for soman, and 10 mg·min/m^3 for VX. [A Ct is the product of the concentration (C) of a vapor or aerosol and the time (t) in which one is exposed to that concentration. Thus, if the concentration is 5 mg/m^3 and a person (or animal) is in that concentration for 10 min, the person is exposed to a Ct of 5 times 10 or 50 mg·min/m^3. This is not an exact measure of dose, as it does not consider minute volume, percentage retention of the compound, and other fractors. The L and $_{50}$ have the same meaning as in LD_{50}, that is, a lethal dose for 50% of the population exposed.] For comparison, the LCt_{50} of hydrogen cyanide, regarded as a highly toxic compound, is 2500 to 5000 mg·min/m^3.

The percutaneous LD_{50}s have been estimated to be 1000 mg for tabun, 1700 mg for sarin, 100 mg for soman, and 10 mg for VX. The disparities between these and the vapor LCt_{50}s have to do with the physicochemical properties of the compounds. The G compounds are much more volatile than VX (and volatility varies greatly among the three) and tend to evaporate from the skin surface rather than penetrate the skin. If evaporation were prevented by occlusion, the LD_{50}s would be more similar, since all these compounds readily penetrate the skin (and normal clothing).

The so called "nerve gases" are clear liquids under temperate conditions, and the preferred term is *nerve agents*. They are are generally odorless, although GA has been reported to have a slight *fruity* odor and GD a slight, nondescript odor. The *G-agents* are volatile under temperate conditions and present a vapor hazard as well as a liquid hazard. Sarin is the most volatile, but is less volatile than water; soman and tabun, in that order, are less volatile. Under temperate conditions (25°C), VX presents a negligible vapor hazard, but its volatility increases as the temperature increases, so at 40 to 45°C, it too would present a vapor hazard.

V. Mechanism of Action

Nerve agents belong to a group of chemicals that exert biological effects by inhibiting the enzyme acetylcholinesterase (AChE). The two major categories of these chemicals are carbamates and OP compounds. Among the former are physostigmine (eserine), neostigmine (used in medicine for several decades), pyridostigmine (used in the therapy of myasthenia gravis, and recently fielded by the military of several countries as a pretreatment or *antidote-enhancing* compound), and dozens of commercially available insecticides (e.g., carbaryl, or Sevin®). The OP compounds include Malathion and dozens of other insecticides. A major difference between the nerve agents and these related compounds is potency. Using human erythrocyte, brain, and muscle ChE, an early *in vitro* study demonstrated that sarin had 10 times more inhibitory activity than TEPP, 30 times more than neostigmine, 100 times more than diisopropyl phosphorofluoridate (DFP; used in pharmacology and formerly used as a therapeutic drug), and 1000 times that of parathion (Grob and Harvey, 1958).

Acetylcholinesterase is a tissue enzyme which catalyzes the hydrolysis of acetylcholine (ACh), the neurotransmitter in the cholinergic portion of the nervous system. It is located at the nicotinic receptor sites (nAChR) and the muscarinic receptor sites (mAChR); these sites are so named because they can be stimulated by nicotine and muscarine respectively. Nicotinic sites include those on skeletal muscle and those at the termination of the preganglionic autonomic fibers. Muscarinic sites include those innervated by postganglionic parasympathetic fibers (which include the glands of the gastrointestinal and respiratory systems), those in the musculature of the gastrointestinal and respiratory systems, and those of the efferent organs of the cranial nerves (e.g., the heart via the vagus nerve).

Acetylcholine, the neurotransmitter, is released by a nerve impulse and diffuses across the synaptic cleft to combine with the receptor site on the postsynaptic membrane to produce a postsynaptic action potential, which initiates activity in the organ innervated. To prevent further postsynaptic action potentials, ACh is rapidly hydrolyzed by AChE, which attaches to the choline

moiety via its anionic site and to the acetyl moiety via its esteratic site. The resulting reaction produces choline, acetic acid, and the regenerated enzyme. If the neurotransmitter could not be destroyed, it would continue to produce postsynaptic action potentials and continuous activity in the organ. Nerve agents and related compounds inhibit the activity of the AChE by attaching to its active sites. Acetylcholine then cannot attach, is not hydrolyzed, and continues to produce action potentials until the mechanism is fatigued. The biological effects of ChE-inhibiting substances are the result of the excess ACh that cannot be hydrolyzed.

Carbamates initially attach to both the esteratic and anionic sites of AChE, but a moiety of the carbamate is immediately split off, leaving the enzyme carbamylated at the esteratic site. Decarbamylation of the enzyme requires minutes to hours (e.g., about an hour for physostigmine, 4–6 hr for pyridostigmine), and during this time, the enzyme is inhibited or inactive. In contrast, most OP compounds combine only at the esteratic site of the enzyme, and the time of dephosphorylation is determined by the structure of the attaching compound. For many OP compounds, cleavage from the enzyme does not occur, and enzymatic activity returns only with the synthesis of new enzyme. This is often referred to as *irreversible* inhibition of the enzyme, and most OP compounds are irreversible inhibitors. In contrast, carbamates are considered to be *reversible* inhibitors, since the enzyme inhibition is soon reversed by decarbamylation.

If one substance, e.g., a carbamate, is attached to the esteratic site, another, e.g., a nerve agent, cannot bind to that same site. The first compound then *protects* the enzyme from the second compound. This is the basis for the use of the carbamate pyridostigmine as a *pretreatment* compound for nerve agent exposure, which is discussed later.

A. Blood Cholinesterases

Other cholinesterases are found in the erythrocyte [erythrocyte, red blood cell (RBC), or *true* ChE] and in the plasma (serum, plasma, pseudo ChE). The latter, also known as butyrocholinesterase (BChE) because of its very high affinity for butyrocholine, is also found in tissue where its physiological function is unclear [although it apparently has a role in the canine tracheal smooth muscle (Adler *et al.*, 1990), the canine ventricular conducting system (Kent *et al.*, 1974), and the rat atria (Slavikova *et al.*, 1982)].

The activity of these *blood* ChEs can be determined in clinical laboratories and is used to monitor those who work around ChE inhibitors, e.g., crop dusters and depot workers, to provide a confirmation of intoxication by an inhibitor, and to follow the recovery of such a patient.

Butyrocholinesterase is synthesized in the liver (and its activity is decreased in diseases of that organ) and has a replacement time of about 50 days. An uncommon familial abnormality in this enzyme renders one very susceptible to the effects of succinylcholine; this condition is best diagnosed by estimating the *dibucaine number* (Kalow and Genest, 1957). Temporal variation in BChE activity is large, with a reported difference of 50% over a year (Sidell and Kaminskis, 1975a). The enzyme activity is lower in women than in men (Sidell and Kaminskis, 1975b; Hayes, 1982), and even lower in women taking oral contraceptives (Sidell and Kaminskis, 1975b; Robertson, 1967; Whittaker *et al.*, 1971).

Erythrocyte ChE is synthesized with the erythrocyte, and after irreversible inhibition, the recovery rate is approximately that of erythrocyte turnover, i.e., about 1% per day. Activity is low in certain diseases of the erythrocyte, e.g., pernicious anemia, and high in reticulocytes (Hayes, 1982). In individuals studied for a year, the activity varied by 11% in men and 16% in women (Sidell and Kaminskis, 1975a).

1. Preferential Inhibition

After a small exposure, inhibitors usually preferentially inhibit one or the other enzyme. In humans DFP inhibited about 90% of the plasma enzyme, but only 20% of the RBC enzyme (Grob *et al.*, 1947; Ketchum *et al.*, 1973). Some pesticides preferentially inhibit the plasma enzyme, e.g., parathion (Rider *et al.*, 1969; Hayes *et al.*, 1964; Edson, 1964), systox (Rider *et al.*, 1969), and malathion (Hayes, 1982), whereas others initially inhibit the RBC enzyme, e.g., dimefox (Edson, 1964), mevinphos (Rider *et al.*, 1975). The nerve agent VX has a much higher affinity for the RBC enzyme in humans, as there was a 70% or greater inhibition in this enzyme, whereas the plasma ChE was inhibited by only 20% (Sidell and Groff, 1974; Sim, 1962). Sarin also preferentially inhibits the cellular enzyme in humans, e.g., 80–100% inhibition of the RBC-ChE with 30 to 50% inhibition of plasma ChE (Grob and Harvey, 1958; Ketchum *et al.*, 1973). This information is useful for occupational monitoring or to confirm a mild or moderate exposure, but after a large exposure, the activity of both blood ChEs is reduced to zero (Sidell, 1974). For monitoring purposes, the ChE to be used must be selected with knowledge of the preferential inhibitory activity of the ChE inhibitor being used.

2. Relation to Clinical Effects

The nerve agents primarily present a hazard by vapor and by liquid (Table I). The former will cause *local* effects in the organs of the unprotected face (eyes, nose) and in the lungs, and systemic effects by inhalation. Liquid will

Table I
Significant Clinical Effects of Nerve Agents in Humans

Eye
Miosis (uni- or bilateral), conjunctival injection; pain in or around eye; complaints of dim vision or blurred vision
Nose
Rhinorrhea
Mouth
Salivation
Pulmonary tract
Bronchoconstriction and secretions, cough; complaints of "tight chest," "shortness of breath;" on exam: wheezing, rales, rhonchi
Gastrointestinal tract
Increase in secretions and motility; nausea, vomiting, diarrhea; complaints of abdominal cramps, pain
Skin and sweat glands
Sweating
Muscular
Fasciculations ("rippling"), local or generalized; twitching of muscle groups, flaccid paralysis; complaints: twitching, weakness
Cardiovascular
Decrease or increase in heart rate; usually increase in blood pressure
Central nervous system
Acute effects of severe exposure
Loss of consciousness, convulsions (or seizures after muscular paralysis), depression of respiratory center to produce apnea, coma
Acute effects of small exposure or lingering effects (days to weeks)
Forgetfulness, irritability, impaired judgment, decreased comprehension, a feeling of "tenseness" or "uneasiness," depression, insomnia, nightmares, difficulties with expression

produce local effects in the organs under the skin (sweat glands, muscle) and systemic effects through percutaneous absorption. After exposure to a small amount of vapor, there is no good relationship between the severity of local effects and the amount of inhibition of blood ChE activity (Harvey, 1952; Craig and Woodson, 1959). These observations were borne out in a retrospective analysis of the records of 62 people seen at an emergency aid station because of one or more physical signs of nerve-agent exposure. As shown in Table II, the RBC-ChE activity varied greatly despite rather mild effects of the exposure.

In general, systemic effects in humans occur when the RBC-ChE is inhibited by 75 to 80% (to 20 to 25% of normal) (Sim, 1962; Sidell and Groff, 1974). In a study of the percutaneous application of VX, 30 subjects were

Table II
Cholinesterase— Relation to Signs and Symptoms

Effect	*N*	Range of RBC-ChE activity (% of baseline)
Miosis (bilateral)	22	0–100
Miosis (unilateral)	7	3–100
Miosis and "tight chest"	12	28–100
Miosis and rhinorrhea	9	5–90
Miosis, rhinorrhea, and "tight chest"	9	20–92
Rhinorrhea and "tight chest"	3	89–90

Table III
Cholinesterase Inhibition and Systemic Effects

Minimal RBC-ChE (% of control)	*N*	Vomited (*n*)	Vomited (%)
Above 50%	166	1	0.6
40–49%	24	2	8
30–39%	27	9	33
20–29%	42	19	45
Under 20%	24	16	67

asymptomatic with RBC-ChEs as low as 40% of control activity. Nine of 21 whose RBC-ChE activity was 30–39% had signs and symptoms, 10 of 14 with RBC-ChE activity between 20 and 29% had signs and symptoms, and 3 of 5 whose enzyme activity was under 19% had signs and symptoms (Sim, 1962). The data in Table III, a compilation from these and other individuals, suggest that systemic effects occur when the RBC-ChE is inhibited to 20% of normal.

3. Time Course of Inhibition

After vapor exposure, maximal inhibition of ChE activity and biological effects occur within minutes or even seconds. After percutaneous exposure to a large amount of agent (an LD_{50} or greater), enzyme inhibition and onset of effects occur within 1 to 30 min, with the time inversely related to the amount of agent. After small amounts of agent, factors such as site on the body and environmental temperature are important. After equipotent amounts of VX were placed on the head or neck, effects occurred 5 hr later; the interval was

7 hr when the agent was placed on the extremities, and 10 hr when it was on the torso (Sim, 1962). In another study, effects did not occur until 18 hr after agent contact (Bowers *et al.*, 1964).

Temperature also affects rate of agent penetration through skin. Agent was applied at environmental temperatures ranging from 0° to 124°F. Three hr later the skin was decontaminated and the subjects taken to a recovery area (about 80°F). The RBC-ChE continued to decline, and maximal inhibition occurred at 5.6 hr (initial temperature of 124°F), 8.5 hr (68°F), 10.4 hr (36°F), and 12.2 hr (0°F) after exposure. The rate of RBC-ChE activity inhibition increased after the subjects were taken from the cold. These data also serve as a reminder that agent continues to penetrate through the skin after it has been decontaminated from the surface of the skin (Craig *et al.*, 1977).

VI. Effects on Organ Systems

The following paragraphs review available information on the effects of nerve agents on several organ systems in humans (Table III). This information is from studies done in the immediate post-World War II period, from evaluation of patients accidentally exposed to nerve agents and related pesticides, and from personal experience. Some areas were studied more intensive than others, and for some there are few human data, e.g., the neuromuscular system.

A. Eyes

The effects of nerve agents on the eye include miosis and conjunctival injection, and the patient may complain of pain in or around the eye, dim vision, or blurred vision.

Eye effects most commonly are the result of direct contact of the eye with vapor or aerosol agent. (Exposure to a Ct of 3 mg·min/m^3 of GB vapor will produce miosis in most of the population.) After exposure to the agent by other routes, e.g., percutaneous, eye effects may not occur, or may have a very delayed onset, and will not be early evidence of exposure even though more severe effects have occurred. A nerve agent was administered percutaneously, intravenously, or orally to large groups of subjects; many had significant effects (vomiting, sweating, weakness), but none had miosis (Sim and Stubbs, 1960; Sim, 1962; Sidell and Groff, 1974). In 47 patients with parathion poisoning, all 14 severely affected people had miosis, but only 6 of 11 moderately affected and 5 of 22 mild patients had this eye sign (Namba *et al.*, 1971).

After vapor exposure, miosis begins within seconds to minutes, but may not be maximal for an hour or so if the concentration is quite small. The duration of miosis varies, depending on the amount of exposure. The pupils

may appear normal in outdoor or bright indoor light within several days, but their ability to dilate maximally in darkness may not return for 4 to 6 weeks (Rengstorff, 1985; Sidell, 1974).

Vapor or aerosol might cause miosis with no other evidence of exposure. As noted earlier, there is not a good correlation between the presence of miosis and blood ChE activity. However, an early study suggested a relationship between degree of miosis and amount (Ct) of exposure (Johns, 1952).

Unilateral miosis can occur and is usually owing to a leak in the eyepiece of a protective mask, or a volatile agent held close to an eye. The RBC-ChE may or may not be inhibited. The patient may have difficulty with depth perception (the Pulfrich stereo effect, discussed in Hayes, 1982), and the patient should be cautioned about this, e.g., when driving.

Dim vision is usually attributed to the decrease in pupil size. The area of the pupillary aperture correlated with decrease in visual sensitivity in one study (Stewart *et al.*, 1968). However, *dim vision* was demonstrated in the absence of miosis when nerve agent was administered systematically (Rubin and Goldberg, 1958), and dim vision was not present when miosis was produced by topical application of nerve agent to the eye (Rubin *et al.*, 1957). The dim vision could be reversed by the systemic administration of atropine, which enters the central nervous system, but not by atropine methylnitrate, which does not; neither drug altered the pupil size (Rubin and Goldberg, 1958). The authors suggested that neural mechanisms, probably mediated by AChE, in the retina or elsewhere in the central nervous system (CNS), contribute to the dim vision. Further evidence for this is that exposed individuals reported that dim vision regressed before the miosis changed (Craig and Freeman, 1953). Dim vision will remain well past the acute crisis and will cause the patient difficulty in dim light (e.g., he should be warned against trying to drive a car in the evening or at night).

Blurred vision is often a complaint, but in one study, individuals who were exposed to nerve-agent vapor had no change or an improvement in both near and far vision (Moylan-Jones and Thomas, 1973). Two presbyopic workers, accidentally exposed to a nerve-agent vapor, had improved vision until the agent effects decreased (Rengstorff, 1985). These and other authors have attributed the lack of significant change or improvement in acuity to the "pin-hole" effect of the small pupils.

Pain in or around the eye is a common complaint after exposure to nerve-agent vapor. It may be mild or severe, and may be localized to the eyeball or diffuse in the surrounding area or throughout the head. This is generally attributed to ciliary spasm and is markedly worsened by a bright light, e.g., the light from a match when lighting a cigarette. Local instillation of an anticholinergic drug (atropine, homatropine) will bring relief, and severe pain is the only indication for the use of a topical anticholinergic, as these drugs produce blurring of vision (Moylan-Jones and Thomas, 1973).

B. Nose

Rhinorrhea is common after exposure to a nerve-agent vapor and may precede miosis as the first indication of exposure. After exposure to only a small amount of agent, the rhinorrhea might be severe, like a "leaking faucet" or "worse than a cold or hay fever," according to patients.

After a severe exposure by any route, rhinorrhea occurs as part of a generalized increase in secretions, and is less noticeable and of less concern.

C. Lungs

A "tight chest" or "shortness of breath" is a common complaint of those exposed to small amounts of nerve-agent vapor, and as the Ct increases, the patient becomes progressively more severely dyspneic. Exposure to a Ct of 5 $mg \cdot min/m^3$ will cause dyspnea in most people. Bronchoconstriction and secretions from the cells of the bronchi, both with muscarinic receptor sites, contribute to this.

After an exposure to a large amount of vapor, respirations become gasping and irregular within minutes and may cease altogether if the amount of agent is great. Apnea also occurs within minutes of onset of effects from a large percutaneous exposure.

The onset time for respiratory distress is seconds to minutes after exposure to vapor. If the Ct is large, the patient will have observable signs of respiratory difficulty including cyanosis and audible pulmonary changes, and relief is obtained only after therapeutic intervention. On the other hand, after a very small exposure, it is not uncommon for the patient to have mild or moderate respiratory discomfort for 10 to 30 min, and then to have the discomfort dissipate over the following minutes. In this author's experience, people arriving at an aid station 15–20 min from the exposure site often reported "I had a lot of trouble breathing for a while, but I'm back to normal now." After percutaneous exposure, the pulmonary changes may occur within minutes or may be delayed for hours.

In addition to contributing to the dyspnea in a breathing patient, secretions may impede attempts at ventilation in an apneic patient. In several instances, thick mucoid plugs hampered ventilatory efforts until they were removed by suction. Atropine, by drying the thinner secretions, may contribute to the formation of this thick mucus.

Death in nerve-agent poisoning is from respiratory failure, which in animals studies occurred before circulatory failure (Wright, 1954; Rickett *et al.*, 1986). The contributing factors are bronchoconstriction and bronchosecretions that produce obstruction in the airways, weakness followed by a flaccid paralysis of the skeletal muscles (including the muscles of respiration), and failure of the central drive for respiration.

The relative contribution of each of these factors is unclear despite numerous studies over the past five decades. The lack of CNS drive was felt to dominate in most species, but there was variation between species and agents (DeCandole *et al.*, 1953). For example, bronchoconstriction was found to be much more severe in the dog than in the monkey (Johnson *et al.*, 1985), but in the rabbit, bronchoconstriction was a minor factor, and neuromuscular block and lack of CNS drive were the primary causes of respiratory failure (Wright, 1954). In a recent study in cats, loss of central drive was the predominant factor, whereas the contribution of bronchoconstriction was insignificant (Rickett *et al.*, 1986).

D. Skeletal Muscle

Skeletal muscle effects of nerve-agent intoxication include fasciculation, twitches (or jerks), and fatigue and paralysis. Generally, these are seen after an overwhelming exposure or as a local response to agent application on the skin.

Fasciculations are the visible contractions of a small number of fibers innervated by a single motor nerve filament, and look like ripples under the skin. They occur as a localized response to a drop of agent on the skin, and after a severe exposure, involve most skeletal muscle. Generalized fasciculations are a very characteristic sign of severe poisoning by this group of agents, and are seen in the acute phase and early in the recovery stage.

Twitches are severe and sudden unsynchronized contractions of large muscle groups, causing the limbs to flail about or momentarily to become rigid. Instead of twitches, sometimes there is prolonged contraction of muscle groups, particularly those groups near the site of exposure, e.g., an individual was exposed to soman near his mouth and had marked trismus and nuchal rigidity (Sidell, 1974).

After a few minutes of hyperactivity, muscles fatigue and become flaccidly paralyzed. Unless there is intervention, this is a terminal stage.

E. Heart

1. Rate

Although it is usually stated that ChE inhibitors cause bradycardia, this is not always true in human exposures. Bradycardia is to be expected in an isolated preparation in which the vagal nerve is the dominant influence on the heart. However, in severe poisoning there is adrenergic stimulation from the preganglionic cholinergic fibers, and there may be fright, hypoxia, and shock. The heart rate is the result of these competing factors.

In a review of the records of 199 patients seen for mild to moderate nerve-agent exposure (who had at least one definite sign of exposure), 13 had a presenting heart rate under 64 beats per minute (bpm), 13 had presenting heart rates of 65 to 69 bpm, 63 had heart rates of 70 to 80 bpm, 41 had heart rates of 81 to 89 bpm, 38 had heart rates between 90 and 99, and in 31, the heart rate was over 100. As a heart rate of 64 to 80 is considered normal for adults (Bellet, 1963), 13 (6.5%) had bradycardia, and 110 (55%) had high heart rates (69, or 35%, had rates over 90 bpm).

The initial heart rates have varied in reported severe insecticide exposures. In 10 severe patients (9 of whom had severe impairment of sensorium), the presenting heart rate was over 100 bpm in 7, and over 90 bpm in the other 3 (Ganendran, 1974). In another report, the heart rates of 3 unconscious patients were slow (1 had cardiac arrest) (Willems *et al.*, 1971). In a comprehensive review of OP insecticide poisoning, 2 unconsious patients were reported to have had heart rates of 100 and 80 bpm. The authors note that "blood pressure and heart rate are increased in the acute stage (p. 481). That the heart rate is an unreliable diagnostic sign was noted by its omission from a comprehensive list of signs and symptoms (Namba *et al.*, 1971).

A heart rate of 90 bpm or greater is sometimes suggested as an indication that adequate atropine has been administered to one intoxicated by a ChE inhibitor. As will be discussed later, the heart rate is of no value as a guide to therapy unless it is below 60 bpm, which suggests that atropine has been administered in inadequate amounts.

2. Rhythm

Disturbances in cardiac rhythm occur after nerve-agent intoxication, and most of those reported have been bradyarrhythmias because of the predominance of the vagus nerve. Heart block (first, second, and third degree), idioventricular rhythm, and premature ventricular complexes have been reported in animals exposed to nerve agents (Oberst *et al.*, 1956; Robineau and Guittin, 1987) and in humans exposed to OP pesticides (Kiss and Fazekas, 1979). The therapeutic drug atropine (2 mg, intramuscularly) can cause transient atrioventricular dissociation and a few minutes of bradycardia before the characteristic tachycardia.

Once tachycardia occurs, the other arrhythmias usually do not reappear and require no specific therapy. However, there are two causes for concern. *Torsade de pointes*, a rapid, multifocal ventricular arrhythmia, has been reported after nerve-agent poisoning in animals (Robineau and Guittin, 1987) and after OP insecticide intoxication in humans (Ludomirsky *et al.*, 1982). The second is ventricular fibrillation, which occurs in nerve-agent-poisoned hypoxic animals when atropine is administered intravenously (Freeman *et al.*, 1954; Kunkel *et al.*, 1973; Wills *et al.*, 1950).

the psychological changes. The onset of signs and symptoms, physical or psychological, occurred between 3 and 18 hr, and physical signs did not always accompany the psychological changes. Illogical or inappropriate trends in language and thinking and perceptual distortion (delusions, hallucinations) were not noted (Bowers *et al.*, 1964).

An individual severely intoxicated with soman exhibited psychological changes (withdrawal, depression, antisocial thoughts) in the days following the acute episode. In a controlled study, he was given scopolamine hydrobromide (a cholinergic blocking compound, with high CNS effectiveness) or scopolamine methylbromide (which does not enter the CNS). On the days he received the hydrobromide salt, he felt better and performed better on a test of cognitive function. This suggested that the centrally active drug blocked the effects of the remaining excess acetylcholine (Sidell, 1974).

These reported observations correspond to personal experience. Mild psychological disturbances are probably more common than usually recognized. Their incidence is higher after severe exposure, but they occur with some regularity in individuals with few or no physical signs of exposure. They begin hours to a day after exposure and may linger from days to many weeks. These people may appear irritable, they are forgetful ("for the first time in 10 years, I forgot my lunch"), they do not answer questions as quickly and precisely as usual, and they may exhibit impaired judgement, poor comprehension, and decreased ability to communicate. Gross mental aberrations, e.g., hallucinations or disorientation, are not part of the picture. These effects must be evaluated before discharging a patient from medical care.

2. Electroencephalogram

Electroencephalographic (EEG) changes after DEP administration to humans included greater variations in potential, increased frequency with increased beta rhythm, more irregularities in rhythm, and the intermittent appearance of abnormal waves. These changes usually followed the onset of CNS symptoms, could be correlated with depression of RBC-ChE (but not plasma ChE) activity, and were decreased or reversed by atropine (Grob *et al.*, 1947). An EEG of a person severely intoxicated by sarin, taken after the loss of consciousness, showed marked slowing with bursts of high-voltage five-per-second waves. These changes persisted for 6 days despite atropine administration (Grob, 1956).

After smaller amounts of sarin, EEG changes correlated with symptoms. Those individuals with mild symptoms had a slight diminution of voltage, and those with moderate symptoms had irregularities in rhythm, variation in potential, and intermittent bursts of abnormal waves. These changes persisted for 4 to 18 days after disappearance of symptoms and were decreased by atropine (Grob and Harvey, 1958).

3. Seizures and Brain Damage

Reviews (Levin and Rodnitzky, 1976; Karczmar, 1984) and case reports (Sidell, 1974) have described longer lasting psychological disturbances caused by OP insecticides and nerve agents. Generally, these lasted for weeks to months, but may remain for years (Duffy *et al.*, 1979). These may relate to findings in animals.

A series of studies in animals indicates that morphologic changes in the brain may occur after nerve-agent intoxication. In soman-poisoned rats (Lemercier *et al.*, 1983; Petras, 1981; McLeod *et al.*, 1984), monkeys (Wall, 1987; Petras, 1984), and baboons (Anzueto *et al.*, 1986), in sarin-poisoned rats (Singer *et al.*, 1987), and in VX-poisoned rats (McDonough *et al.*, 1987), neuronal degeneration and necrosis were seen on necropsy as long as 45 weeks after exposure. Hypoxia was suggested as the etiological factor, and in rats with bicuculline-maintained convulsions, there is evidence both for (Blennow *et al.*, 1978) and against (Soderfeldt *et al.*, 1983) this hypothesis.

In most instances there was a direct correlation between convulsive activity and brain damage, and between duration and severity of convulsive activity and severity of brain damage (although in several studies the investigators noted the lack of such activity). Brain damage after prolonged convulsive activity has been known since 1880 (Meldrum, 1983) and occurs after convulsions induced by other compounds, e.g., in rats after fluorothyl (Nevander *et al.*, 1985), and in baboons after bicuculline (Meldrum and Brierly, 1973). The mortality in humans with prolonged convulsions (over 30 min) was reported to be 6–30%, but twice that number have irreversible neurological deficits after the convulsive activity (Orlowski *et al.*, 1984).

Animals surviving severe soman intoxication had decrements in performance, as measured on a variety of behavioral tests, which lasted until sacrifice at 4 months (Raffaele *et al.*, 1987; McDonough *et al.*, 1986; Modrow and Jaax, 1987).

Diazepam, an anticonvulsant of the benzodiazepene family, was shown to control soman-induced convulsions in monkeys (Lipp, 1973) and other ChE inhibitor-induced convulsions in the rabbit (Rump *et al.*, 1972, 1973). In soman-poisoned rats, it decreased convulsions and brain damage (although without atropine, it did not decrease mortality) (Martin *et al.*, 1985). When given with 2-PAMCl, with or without atropine, diazepam reduced the severity but not the incidence of brain lesions in soman-poisoned rats (McDonough *et al.*, 1989). In monkeys, pretreated with pyridostigmine and challenged with soman followed by atropine and 2-PAMCl therapy, diazepam decreased the morphologic brain lesions in most areas of the brain, although those in the frontal cortex were increased (Hayward *et al.*, 1988). It has also been useful in ameliorating ChE-inhibiting insecticide-induced convulsions in humans.

At the USAF School of Aerospace Medicine, monkeys were trained to operate the Primate Equilibrium Platform (PEP), an apparatus designed to

gauge complex cerebellar-cortical function and fine motor control. After receiving several lethal doses of soman, the animals were all treated with atropine and 2-PAMCl, and some were given diazepam and some were not. Although the incidence of convulsions in the two groups was similar, those receiving diazepam convulsed for 11 min (mean), whereas the others convulsed for 98 min (mean). Those receiving diazepam performed much better on the PEP over the next 14 days (Blick *et al.*, 1989), and had less brain damage (one of eight survivors of the diazepam group had severe lesions, versus three of four non-diazepam survivors) (Switzer *et al.*, 1990).

Information from the few severe accidental human exposures that have been reported indicates that after a large dose, a person loses consciousness within seconds and has convulsive activity for several minutes, until it is terminated by cessation of breathing and flaccid paralysis. In these cases, medical intervention has prevented death, but it seems likely that had there been no intervention, death would have occurred. In no case was there prolonged convulsive activity, and possibly this would not occur because of the intervention of medical care or death. However, in at least one instance there was a suggestion of prolonged (4–6 weeks) mental impairment despite minimal convulsive activity (Sidell, 1974).

Pyridostigmine pretreatment (described in a later section) permits the survival of animals receiving many times the untreated LD_{50} of soman. The animals maintain ventilation and continue to have convulsive activity for long periods. Under these circumstances the incidence of brain damage will be high unless the convulsions are stopped or ameliorated, e.g., with diazepam.

VII. Clinical Intoxication

After a large amount of nerve agent by any route of exposure, the clinical effects are preciptate in onset and catastrophic in magnitude. In most instances in which people have been accidentally exposed to these compounds, the exposure has been of less magnitude and the response less severe, and most scenerios of anticipated exposure of groups of people suggest many mildly to moderately exposed individuals for each severe casualty. The initial effects or the presenting signs and symptoms depend on the route and amount of exposure. The two most common routes of exposure are to vapor and to liquid (percutaneous).

A. Vapor Exposure

After exposure of an unprotected person to vapor, local effects are followed by more severe, systemic disturbances (Table IV). The initial effects of a small

Table IV.
Effects of Vapor Exposure to Nerve Agents[a]

Exposure to small amount (local effects)
Miosis
Rhinorrhea
Slight bronchoconstriction/secretions (slight dyspnea)
Exposure to moderate amount (local effects)
Miosis
Rhinorrhea
Bronchoconstriction/secretions (moderate to marked dyspnea)
Exposure to large amount
As above plus:
Loss of consciousness
Convulsions (seizures)
Generalized fasciculations
Flaccid paralysis
Apnea
Involuntary micturition/defecation

[a] Onset within seconds to several minutes after onset of exposure.

amount of the agent are on the eyes, the nose, and the lungs. Involvement of one or more of these three organs is quite characteristic. Table II indicates the approximate incidence of involvement of these organs, singly or in combination, in an unselected group. These are local effects, or direct effects of the agent on the organ, and do not indicate systemic absorption of the agent (see section on ChE inhibition).

Eye effects are often the first to appear, since the eye is quite sensitive to these agents (the Ct_{50} of sarin to cause miosis is approximately 3 mg·min/m^3). Dim vision is commonly the first symptom noted, particularly by those with previous exposure who quickly recognize this. The miosis and possibly conjunctival injection will be recognized by an observer. Rhinorrhea, which commonly accompanies this, may be mild or quite severe ("it was filling my mask"). Discomfort in breathing ("my chest is tight"), without objective signs of pulmonary involvment, may also be noted. However, each of this triad may occur without the others.

Further exposure causes an increase in secretions from the nose and the salivary glands, and an increase in pulmonary manifestations. Respiratory distress, accompanied by physical signs of bronchoconstriction and secretions, will be obvious to the observer. The patient will be gasping, and other objective signs of respiratory impairment (e.g., mild cyanosis) may be present.

Following an overwhelming exposure, the patient may lose consciousness within seconds, and develop convulsive activity within a minute or two. After

several minutes, respiratory arrest and flaccid paralysis intervene, and death will shortly follow without intervention. On examination, the patient will be unconscious or postictal, and will display miosis, copious secretions from the nose and mouth, apnea or labored and gasping irregular respiratory efforts, muscular twitching and generalized muscular fasciculations (a characteristic finding that may continue into the recovery phase), or a flaccid paralysis, and possibly evidence of involuntary defecation or micturition.

In instances of severe vapor exposure, the onset has been precipitate after as little as one breath of the vapor. One individual later recalled he noted an increase in secretions, and another reported feeling "giddy" or "faint" before losing consciouness. Neither had time to react, and both were unconscious within a minute of exposure.

After vapor exposure the biological response begins within seconds, and unless the concentration is quite small, reaches maximal intensity within minutes. After vapor exposure ceases (removal from the area, masking) the response does not significantly increase.

B. Percutaneous Exposure (Liquid)

Percutaneous exposure differs from vapor exposure in several important aspects. The local effects may be insignificant and commonly are not noticed. Even after a lethal amount on the skin, there is usually a delay in onset of response. The onset of effects may be hours after removal of agent from the skin (decontamination), and the response may continue to worsen once begun (Table V).

A small amount of liquid percutaneously causes the local responses of increased sweating and muscular fasciculations around the site. Unless one knows of the exposure, these are usually not noticed. Later, systemic effects may follow and consist of gastroinstestinal signs and symptoms (nausea, vomiting, diarrhea) and a feeling of "tiredness" or "weakness," sometimes accompanied by muscular twitching or fasciculations, or by psychological manifestations. The onset of these may be as long as 18 hr after exposure and has also been reported 3 hr after decontamination (see earlier section of time course of ChE inhibition). The response may increase as the agent continues to be absorbed, with moderate to severe respiratory distress and more severe muscular manifestations following.

In several instances of severe exposure, an asymptomatic period of 10 to 30 min preceded a precipitant onset of loss of consciousness, convulsive activity, and cessation of respiration minutes later (e.g., Sidell, 1974). Before

Table V
Effects of Dermal Exposure to Nerve Agents

Minimal exposure
Increased sweating at site of exposure
Muscular fasciculations at site of exposure
Moderate exposure
Increased sweating at site
Muscular fasciculations at site
Nausea, vomiting, and diarrhea
Feeling of generalized weakness
May be precipitant in onset after long (4–18 hr) asymptomatic interval
Severe exposure
Above may be present
Loss of consciousness (may be precipitous in onset after asymptomatic interval)
Convulsions (seizures)
Generalized fasciculations
Flaccid paralysis
Apnea
Involuntary micturition/defecation

apnea and paralysis occur, this is accompanied by miosis, increased secretions, muscular twitching and fasciculations, and possibly involuntary micturition and defecation.

VIII. Medical Management

The principles of care for a nerve agent-intoxicated person are the same as those for any toxic substance exposure, namely terminate the exposure, maintain ventilation and circulation, and administer antidotes.

Often overlooked is the need for the medical care provider to protect himself. If contaminated, he will become an additional casualty. This advice seems obvious, yet as recently as the Iran–Iraq conflict, there were stories that contaminated casualties were sent to Europe for medical care, and the care providers (not wearing protective apparel) who received them suffered effects from agent remaining on the patients or on their clothing.

Protection can be achieved by wearing protective apparel (an appropriate protective mask, heavy rubber gloves, heavy rubber apron, or encapsulation) or by ensuring that the casualty is completely decontaminated.

Whether first to terminate the exposure, establish ventilation, or to administer antidotes will depend on the condition of the patient and facilities

available. Their order in the following does not necessarily suggest the order in which they should be undertaken.

A. Terminating the Exposure

If the harzard is from vapor alone (i.e., no possible contact with liquid agent), termination of the exposure is as simple as removing the patient to a toxic-vapor-free environment. If the agent is in a room or building, removal of the patient from that structure may be all that is necessary. If outside, this requires knowledge of the magnitude of the agent cloud and wind direction, and the distance to clean air might be longer than can be traversed quickly. An alternative is to put a protective mask on the patient, but this could interfere with ventilatory support if this is needed. In the military, the M17A2 mask provides considerable protection if properly fitted and sealed, and in the civilian community, a Self-Contained Breathing Apparatus (SCBA) or another device would be adequate. These may not always be available on the site, and there are difficulties in fitting them to a struggling patient.

Terminating the exposure usually involves removing liquid agent from skin and clothing, or decontamination. Clothing should be removed as early as possible. If the site is well localized, that section should be cut away leaving wide margins, but if the extent of the contamination is unknown, all clothing should be removed and the underlying skin thoroughly decontaminated. Clothing offers negligible protection from nerve agents.

Decontamination may be accomplished by physical removal or by neutralization of the agent. When droplets can be seen, they can be blotted off (care should be used, because scraping or wiping abrade the skin, enhancing agent penetration) followed by flushing with water, or rinsed off with copious amounts of water. The agents can be neutralized with alkaline solutions (soap and water) or chlorine-releasing substances (e.g., household bleach), but these solutions may damage the skin (current policy in the military is to use 0.5% hypochlorite instead of bleach, which is 5%, but in a military setting, a further rinse with water may not be possible). These should be followed with water. Neutralization is not immediate and may take an hour or longer for completion.

Personnel in the military use the M258A1 kit for personal decontamination. This contains sets of two towelettes, one containing a hydroxide and phenol and the other chloramine, a chlorine-releasing compound.

Most civilian emergency medical facilities are not equipped for the entry of contaminated patients. An appropriate facility will have a negative-pressure, filtered-air system to reduce the vapor hazard for the staff and the rest of the building, and a drainage system to contain the contaminated washoff produced during the decontamination procedure. It will also have an adequate

supply of protective clothing for those who perform decontamination of the patient and for the medical care providers who treat the patient before decontamination.

Decontamination is performed to reduce further absorption of the agent as well as its spread to unexposed areas of the casualty, and to prevent spread of the agent to others who handle the patient.

B. Ventilation

Respiratory embarrassment is a feature of all but the mildest exposures to nerve agents. In conscious patients who have received a small to moderate amount of agent, administration of the antidote(s) will usually reverse the bronchoconstriction, reduce the excess secretions, and provide relief within minutes. Ventilatory support is seldom required under these circumstances, although if the patient is known to have cardiac or pulmonary disease or is elderly, supplemental oxygen may be beneficial.

Inadequate ventilatory efforts followed by apnea occur after a large exposure, and the antidotes are seldom totally effective in reversing this. After endotracheal intubation, assisted ventilation (preferably with oxygen) must be started and continued until the patient has adequate spontaneous respiration (which in reported cases has ranged from 0.5 to 3 hr). In the absence of personnel skilled in entubation, ventilation must be undertaken by whatever means are available.

Airway resistance is initially high (50–70 cm H_2O) because of the constriction of the airway musculature and copious secretions, and initial attempts at ventilation may be only partially successful. Atropine will reverse these effects, once adequate amounts are given. However, in at least one case, the administration of atropine seemed to thicken the secretions, and attempts at ventilation were unsuccessful until the mucoid plugs were suctioned from the airways.

In the absence of a ventilator, mouth-to-mouth ventilation might be considered. In a toxic environment (vapor), the rescuer will be at risk, and there is also the risk that the patient has contamination on his face. A smaller risk is the expired air from the patient, as less than 10% of inhaled nerve agent is exhaled, most immediately after exposure.

The Schafer method of assisted ventilation was once used briefly in a severe casualty, but even in an individual with normal airways, it provides less than optimal air exchange.

In the severe patient from nerve-agent intoxication, impairment of respiration or total apnea will occur. Although the antidotes will reverse mild or moderate impairment, they are of little benefit against apnea, and assisted ventilation is required for survival.

C. Atropine

Atropine is the drug of choice for intoxication by a ChE-inhibiting substance. Although a second drug, an oxime, provides synergistic benefit against most of these substances, the use of atropine alone (with ventilation) will support survival in most cases.

Atropine blocks the effects of excess ACh and protects the receptor from further stimulation. Atropine is most effective at muscarinic receptor sites and has minimal effects at nicotinic receptor sites.

The mutual antagonism of a ChE-inhibiting compound and a cholinergic blocking compound was first recognized over a century ago (Argyll-Robertson, 1863). In the past five decades, many cholinergic blocking compounds were studied as antidotes to nerve agents. In general, almost any compound with cholinergic blocking activity has antidotal activity. Those with higher lipoid solubility (better penetration into the CNS) have some benefits, but also have more side effects if administered erroneously or too aggressively to someone with a mild exposure.

In the post-World War II period, atropine was selected because it was effective and because its side effects were tolerable. In the same period, the military selected the dose of 2 mg for an injector for self or buddy use by military personnel. It was recognized that the selection was a compromise between the therapeutically desirable dose and an amount that could be administered (self or buddy) to a nonintoxicated individual safely and without causing damaging performance decrements.

The side effects of 2 mg of atropine in a normal young person (without nerve-agent exposure) are an increase in heart rate (of about 35 bpm, which usually is not noticed by the recipient), drying of secretions (those in the mouth are most noticeable), decrease in sweating, mydriasis, and paralysis of accommodation. Most effects dissipate in 4 to 6 hr, but visual blurring may continue for 24 hr or longer. A potentially serious effect in non-nerve agent-poisoned individuals is inhibition of sweating, which is hazardous if the temperature is elevated, and the individual is performing work. Thirty-five soldiers successfully hiked for 115 min at 3.3 miles per hour in 83°F weather, but on another day at the same temperature, after receiving 2 mg of atropine, more than half did not complete the march because of core temperatures of 103.5°F (Robinson *et al.*, 1953).

When administered intravenously, atropine (2 mg) will produce a maximal effect on the heart rate in about 3 to 5 min. and other effects follow several minutes later. The use of the Atropen® injector (the device used by the military; Survival Technology Inc., Bethesda, Maryland) for intramuscular atropine administration enhances the rate of absorption, and maximal tachycardia is seen at 34 min (versus 41 min by conventional administration) (Sidell *et al.*, 1974).

Atropine is effective at those sites with muscarinic receptors. After its use, secretions will dry, constriction of smooth muscle will be reversed, and the clinical signs and symptoms caused by abnormalities at these sites will be reduced. Atropine has negligible effects at sites with nicotinic receptors; skeletal muscle twitching and fasciculations may continue long after the patient is otherwise recovering, and their continued presence is not an indication for further atropine. Similarly, systemic atropine, unless given in large amounts, will not reverse miosis, and this eye sign should not be used as an indication for further atropine administration.

Atropine should be started as soon as possible after onset of agent effects, and the initial dose should be at least 2 mg and as much as 6 mg or more. Intravenous administration should be avoided if the patient is hypoxic (risk of ventricular fibrillation), but after ventilation is undertaken, this is an excellent route. If the patient is hypotensive, atropine administration intratracheally or into an endotracheal airway (if one is in place) should be considered for more rapid absorption by the peribronchial vessels. In a mildly or moderately symptomatic patient, an interval of 5 to 10 min between doses is acceptable, and usually a total of 2 to 4 mg will be adequate. In a severely intoxicated person, more frequent administration is needed initially.

In a handful of cases, the total atropine dose in severe patients was in the range of 5 to 20 mg. In contrast, people with severe OP insecticide poisoning have required 500–1000 mg daily for a week or longer. An intravenous drip of atropine has been used in some cases because of the necessity for frequent repeated atropine administration over a long period in pesticide poisoning (LeBlanc *et al.*, 1986). Pesticides cause continuing episodes of acute cholinergic crisis that may continue for days to weeks, possibly because they are sequestered in depots in the body and/or are metabolized more slowly. In contrast, once the acute crisis is over, which is usually a matter of hours, patients with nerve-agent intoxication continue to recover with minimal recurrent evidence of cholinergic stimulation. These differences must be recognized and understood.

Atropine should be continued until secretions are minimized and until ventilation is adequate. For reasons discussed earlier, the heart rate is not a reliable indication of the adequacy of atropine administration, except that bradycardia suggests additional atropine is indicated. A conscious person will indicate comfort in breathing; in an unconscious person, this can be gauged by ease of ventilating.

In general, it is better to err on the side of giving too much atropine rather than too little. If the diagnosis is in error, an overdose of atropine will cause transient side effects, but too little atropine will allow the unantagonized effects of nerve agent to continue unabated.

D. Pralidoxime

Oximes greatly enhance the therapeutic activity of atropine against poisoning by many ChE inhibitors, but are not effective when administered alone. The oxime in use in the United States is 2-PAMCl (pyridine-2-aldoxime methochloride; pralidoxime chloride; Protopam® chloride). Other oximes are preferred by other countries, e.g., P_2S by the United Kingdom.

In the early 1950s, Wilson and associates (Wilson, 1951; Wilson and Ginsburg, 1955) found several types of compounds that would remove the inhibitor from ChE much faster than spontaneous hydrolysis by water. Based on research in both military and civilian laboratories, 2-PAMCl was selected for use.

The pharmacological action of oximes is to remove the OP compound from the inhibited enzyme, but there are limitations to the therapeutic benefit of this. After the OP compound attaches to the enzyme, the inhibitor–enzyme complex may become resistant to cleavage or hydrolysis by certain compounds, a process known as *aging*. The rates of spontaneous hydrolysis and aging depend on the structure of the inhibitor (a more detailed discussion of this process is beyond the scope of this chapter; the interested reader should see, e.g., Koelle, 1975). The clinical importance is that oximes are relatively ineffective after aging occurs.

The VX-inhibited RBC-ChE complex spontaneously reactivates at about 0.5 to 1.0% per hour for the first 48 hr and ages very little during this period (Sim and Stubbs, 1960; Sim, 1962; Sidell and Groff, 1974). Tabun also has a long half-time for aging, about 46 hr (deJong and Waring, 1978). About 5% of a sarin-RBC-ChE complex spontaneously reactivates, and the half-time for aging is about 5 hr (Sidell and Groff, 1974). The soman–enzyme complex does not spontaneously reactivate, and ages in about 2 min (Harris *et al.*, 1978). For this reason, oximes contribute little to the therapy of soman intoxication. Clinically, oximes do not noticeably reverse effects in organs with muscarinic receptors, but do decrease abnormalities in organs with nicotinic receptors (e.g., skeletal muscle).

The recommended dose of 2-PAMCl is 15–25 mg/kg, but the required dose depends on the inhibitor and the interval between poisoning and administration. A concentration of 4 mcg/ml was found to reverse the sarin neuromuscular block in cats (Sundwall, 1961), and a plasma concentration of 6.5 mcg/ml can be achieved by 600 mg given intramuscularly by the ComboPen®, an autoinjector used by the military (Sidell *et al.*, 1974). The oxime 2-PAMCl, 15–20 mg/kg intravenously, reactivated over 50% of the inhibited RBC-ChE 3 hr after sarin administration (Sidell and Groff, 1974). In animal studies, the protective ratio (PR; the ratio of the LD_{50} with a specific treatment to the LD_{50} with another treatment or no treatment) increased from 25 to 90 in sarin-poisoned rabbits when the dose of

intravenous oxime was increased from 5 to 10 mg/kg (O'Leary *et al.*, 1961); it changed from 1.6 to 4.2 when the dose of intramuscular 2-PAMCl was raised from 30 to 120 mg/kg in sarin-poisoned rats (Davies *et al.*, 1958); and it increased from 1.9 to 3.1 after intramuscular 2-PAMCl was increased from 11.2 to 22.5 mg/kg in VX-poisoned rabbits (Sidell *et al.*, 1968). In the first two studies, therapy was administered immediately after agent, and in the third, at the onset of signs. No ventilation was used.

The oxime should be given intravenously and is commercially available in vials containing 1 g cryodessicated 2-PAMCl for this purpose. Slow administration, over 20 min, will minimize the hypertension that may occur at doses above 15 mg/kg or after rapid administration (Calesnick *et al.*, 1967). Hypertension can be quickly but transiently reversed by phentolamine (5 mg, i.v.) (Fig. 1). Other side effects, which may occur at much lower doses (2.5–10 mg/kg), include dizziness, blurred vision, diplopia, and nausea and vomiting, but these are insignificant in a seriously poisoned person.

A solution for intramuscular use can be made by mixing the contents of a 1-g vial with 3 ml sterile water or saline. This produces a concentration of 300 mg/ml, which is what is in the autoinjector (600 mg/2 ml) for the military. A plasma concentration of 4 mcg/ml occurs at 7 min after autoinjector administration (versus 10 min by conventional administration) and a maximal

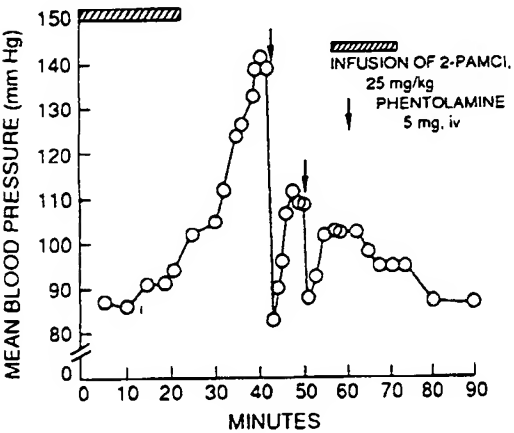


Figure 1 An infusion of 25 mg/kg of 2-PAMCl over 25 min produced marked hypertension, which was rapidly, but transiently reversed by phentolamine, 5 mg. The mean blood pressure is the diastolic plus one third of the difference between the systolic and diastolic.

concentration occurs at 19 (autoinjector) or 22 min (conventional) (Sidell *et al.*, 1974). Doses of 2.5 to 30 mg/kg, i.m., in normal subjects caused no signs or symptoms except pain at the site and mild blood pressure elevation (Sidell and Groff, 1971; Swartz and Sidell, 1974).

The drug is rapidly and rather completely excreted intact in the urine (80–90% within 3 hr) and has a plasma half-life of about an hour (Sidell and Groff, 1971). Its renal clearance is close to that of para-aminohippurate, suggesting tubular secretion (Swartz and Sidell, 1974), and is decreased by heat and/or exercise (Swartz and Sidell, 1973), and by thiamine, which prolongs the half-life and increases the plasma concentration of the oxime (Swartz and Sidell, 1974; Josselson and Sidell, 1977, 1978). Because of its side effects (particularly hypertension) and half-life, repeated administration of 2-PAMCl should be at hourly intervals.

A tablet of 0.5 g 2-PAMCl was commercially available, but the relative slowness of absorption of the drug by this route and the high dose required (5 g, or 10 tablets) limited its use (Sidell *et al.*, 1969).

Although the rapid aging of the soman–ChE complex suggests that 2-PAMCl is of no value in soman poisoning, animal studies suggest it is of some benefit (J. Fleisher, personal communication, 1970; J. von Bredow, personal communication, 1989). It causes a small (5–10%) reactivation of the inhibited blood enzyme (J. von Bredow, personal communication, 1989), and possibly acts as a cholinergic blocking agent at the nicotinic sites (J. Fleisher, personal communication, 1970) or improves circulation by stimulating release of catecholamines (J. von Bredow, personal communication, 1989).

With a few exceptions, oximes are generally not beneficial in carbamate intoxication, and may intensify the effects of the poison with some carbamates, e.g., carbaryl (Sevin®). The manufacturer's brochure should be consulted.

E. Specific Problems

1. Seizures

As detailed earlier, severe poisoning from nerve agents and related substances causes seizure activity, and prolonged seizure activity is associated with morphologic brain damage and performance decrements, at least in animals. Because of this, the U. S. military (and the military forces of other countries) has fielded an autoinjector containing diazepam for buddy or medical use.

Diazepam should be administered to a patient with seizure activity, and also should be administered to those in the *pre-convulsive* phase. This may be defined as someone who has moderate or severe signs of poisoning in more than one organ system (e.g., respiratory and gastrointestinal systems). This patient should also receive at least 6 mg atropine as an initial dose (see the

following). The dose of diazepam is 10 mg, i.m., (or a smaller amount, 2–5 mg, i.v.) initially, with additional doses as required.

2. Cardiac Arrhythmias

Usually, undesirable cardiac arrhythmias terminate when the atropine-induced sinus tachycardia begins. The danger of i.v. administration of atropine in a hypoxic patient has been mentioned.

A young patient poisoned with an insecticide had persistent ST wave elevation and T wave flattening on her electrocardiogram (EKG), felt to be owing to persistent tachycardia caused by large amounts of atropine. After her heart was pharmacologically *isolated* with propranolol, a beta-adrenergic blocking drug, her heart slowed to 107 bpm and her EKG normalized despite further large amounts of atropine (Valero and Golan, 1967). This drug might be useful under these circumstances.

3. Eyes

The temptation to treat miotic pupils with i.m. or i.v. atropine, particularly in a patient with no or few other signs of exposure, must be tempered by the knowledge that the amount of atropine needed to reverse this effect will cause undesirable side effects, even in a healthy young person.

Miosis can be reversed by the topical instillation of ophthalmic preparation of atropine, homatropine, or other drugs (Moylan-Jones and Thomas, 1973), but this will produce blurred vision for a day or more. Except in dim light, vision is not noticeably impaired with miosis. The only indication for topical therapy is intractable pain in or around the eye, for which topical therapy will supply relief.

During the therapy of systemic effects, the adequacy of atropine dosage should not be judged by dilatation of miotic pupils. The amount of secretory activity and ease of ventilation are the proper indicators for atropine administration.

F. Pretreatment

A previous chapter presented the data for the advantages of administering a carbamate before exposure to a nerve agent, and an earlier section in this chapter briefly outlined the molecular basis for this. Studies on physostigmine (described in the previous chapter) demonstrated its effectiveness, but this drug has two disadvantages for human use. At the doses required to produce the desired degree of RBC-ChE inhibition, it caused side effects (D'Mello and Sidell, 1991), and the half-time of the drug in humans is rapid (about an hour),

which would necessitate very frequent dosing (*sustained release* oral preparations and other methods of producing a prolonged absorption have proven unsuccessful). Pyridostigmine was developed by the military (in the United States and other nations) for use against the threat of soman exposure. Preadministration of pyridostigmine to animals challenged with soman and treated with atropine and 2-PAMCl permitted them to survive much larger challenges of soman than they could without the pretreatment (see previous chapter).

Pyridostigmine alone, without atropine and 2-PAMCl therapy after agent challenge, is ineffective; i.e., it does not change the LD_{50} of the agent. Pyridostigmine given after agent challenge is likewise of no benefit; i.e., it is not an antidote. Pyridostigmine administered before agent challenge does not reduce the effects of the agent. Most important, there is no evidence that pyridostigmine is of any benefit against nerve agents with slower aging times; i.e., when used before sarin or VX challenge, it does not notably change the LD_{50} compared to that resulting from the use of therapy alone (Koplovitz *et al.*, 1991). It should be used only when the threat is soman.

G. *Therapeutic Guidelines*

The goals of therapy are to minimize the patient's discomfort and distress, and to stop or reverse the disease process. Discomfort in a conscious patient might be dyspnea, nausea and vomiting, or eye pain. To relieve distress might include terminating convulsive activity and restoring adequate oxygenation. Stopping or reversing the disease process includes correcting these and other abnormalities.

Goals must be realistic. Current drugs will not immediately restore consciousness and spontaneous respiration, nor will they immediately reverse skeletal muscle abnormalities (the twitches and fasciculations may continue long after the patient is conscious, breathing adequately, and otherwise in control of the muscular system). It is futile to give increasing amounts of the antidotes in anticipation of immediate reversal of all functions. Adequacy of ventilation is the most important goal; when this is maintained, normal function in other organs will gradually return.

Analysis of blood for ChE activity is useful for occupational monitoring, but in an exposed patient, one treats the patient, not the ChE activity. If facilities are available, this analysis should be done to document the exposure and to monitor the recovery process (including the effectiveness of the enzyme reactivator). Individuals at risk from occupational exposure to ChE inhibitors (e.g., orchard workers, crop dusters) undergo frequent monitoring and their *normal* or *baseline* value is a matter of record; when they are potentially

exposed, but asymptomatic, their ChE activity might provide evidence of absorption. In an *unbaselined* population, drawing blood for this purpose is of less value because there is a large interindividual range of normal enzyme activity, and it usually takes several days to obtain the results of the analysis.

In the care of any type of chemical agent casualty, the foremost concern of the medical care provider must be to protect oneself (whether by protective apparel or by ensuring that the casualty has been thoroughly decontaminated).

1. Suspected

The management of an asymptomatic person who has been in an area of agent contamination is a matter of judgement. If it is certain that the potential exposure was by vapor alone (the person was some distance from a liquid spill, but possibly was in the vapor cloud), signs and symptoms will appear quickly if the person was in contact with the agent. Because effects from vapor occur within seconds to minutes, if these are not present by the time medical assistance arrives, they most likely will not occur, and the individual needs no further medical attention.

An asymptomatic person who has had liquid agent on his clothing or skin should be decontaminated and observed. Minimal decontamination should include cutting away or removing the clothing in the area of contamination and decontaminating the skin underneath. Stripping the person and thoroughly decontaminating the skin is a more certain means of removing all contamination. Since the onset of effects has been reported as long as 18 hr after exposure, observation should be continued for this period.

2. Mild

A person with miosis (and other eye effects) and/or rhinorrhea will generally not require the antidotes unless the rhinorrhea is severe and distressing or the miosis is causing severe pain. The former requires systemic atropine (2 mg should be adequate); the latter, a topical drug (but see comments, earlier).

When dyspnea is present from vapor only (no possibility of liquid exposure), the initial dose of antidotes should match the severity of the discomfort. If the exposure was small, the dyspnea might be improving or almost resolved 20–30 min after the exposure when medical personnel arrive. Whether to administer the antidote will depend on the patient's discomfort and rate of spontaneous recovery and one's ability to observe the patient for worsening of the effects over the next 10–15 min, although the occurrence of this is very unlikely. Those with mild to moderate dyspnea, which is not resolving, should be given 2–4 mg atropine. An interval of 5 to 10 min should intercede before further drug administration, and usually in a 10-min

interval, there will be improvement (sooner if the initial atropine is given i.v.). A patient with severe dyspnea and distress should be treated with 4 to 6 mg atropine initially. Atropine is very effective in reversing bronchoconstriction and secretions, and if there are no more serious consequences of the vapor exposure, this amount will be adequate. In the military, injectors containing the oxime 2-PAMCl are packaged with the atropine injectors, and instructions are to administer one with each of the first three atropine injectors. Where these are not available, 2-PAMCl (1 g) should be given very slowly intravenously to an individual with definite signs of exposure to the agent.

If the patient is seen within 5 min of vapor exposure (self or buddy aid), therapy should be more aggressive, and 2 mg atropine should be administered for any signs of agent effects, 4 mg if any dyspnea is present, and 6 mg for moderate to severe dyspnea. Although effects from agent vapor exposure occur quickly, they may increase in severity beyond the first few minutes, and the added therapy is in anticipation of this progression.

In contrast to events in a vapor-exposed patient, the progression is more difficult to predict in one exposed to liquid. Signs and symptoms might begin as long as 18 hr after contact with agent, and even 3 hr after decontamination of the skin. For these reasons, the observation of sweating or muscular fasciculations at the known site of agent contact usually portends more severe consequences.

The amount of initial therapy after contact to liquid is also guided in part by the interval between exposure and onset of effects. Gastrointestinal symptoms beginning 6 hr after exposure (and initial decontamination) can be reversed by 2 mg atropine, or at most by an additional 2 mg if the initial dose does not cause marked relief. If these symptoms begin earlier, i.e., an hour or so after exposure, 4 mg should be given initially. Again, 2-PAMCl should be administered.

3. Moderate

A patient with moderate involvement of several organ systems, i.e., respiratory distress, gastrointestinal symptoms and signs, and/or muscular twitching, should always be given 6 mg atropine initially (with 2-PAMCl), whatever the route of exposure or time of onset. Diazepam should also be administered even in the absence of seizure activity. If contact was to vapor only, this should be adequate therapy, although rarely additional atropine might be required. If exposure was to liquid, drug administration should be more aggressive, and additional atropine should be given if there is no definite improvement within 5 to 10 min. Again, the guidelines for adequate atropine administration are drying secretions (which will be copious in all but the mildest exposures) and adequacy of ventilation. Recovery is almost certain when there is no seizure activity, respiration does not stop, and consciousness is not lost.

of a serious regression is negligible (in contrast to poisoning by an insecticide, in which serious cholinergic crises occur repeatedly for days to weeks).

I. Return to Duty

Return to work or to duty depends on the requirements of the job, the necessity for return, and visual abilities and mental status. If the job does not require strenuous physical exertion, fine visual discrimination, or critical mental judgements, the patient might return within a week or two. These factors must be evaluated before sending the patient back. In particular, a careful mental status examination must be performed to detect subtle decrements if the job requires an intact cognitive capability and rapid decision making. An air traffic controller might not return for several months because of slow or poor judgment and difficulty in visualizing and following lights on a screen.

After repeated mild exposures, workers in a laboratory/depot area stopped seeking medical assistance after they developed miosis and rhinorrhea. They preferred to continue working rather than undergo the administrative processes, and they continued to function satisfactorily, although their jobs did not require decision making and fine visual abilities. Soldiers with miosis, rhinorrhea, and mild dyspnea performed satisfactorily (although suboptimally) in a field exercise during the day, but did not do well at night.

A necessity for an individual to return to the job sooner than medically desirable is usually not present in the civilian world. However, in a battle zone a medical unit under attack might require the return to duty of any casualty who can walk and use a weapon. After several days of recovery, most nerve agent casualties, even those who had sustained severe exposures, could perform, although their performance might be less than optimal.

IX. Conclusion

Nerve agents are extremely toxic chemicals capable of causing death within minutes of exposure. Therapy can prevent lethality, but such assistance must be early and intense. Because of pretreatment, self-help, and buddy help, most battlefield casualties will survive.

References

Adler, M., Reutter, S. A., Moore, D. H., and Filbert, M. A. (1990). Regulation of acetylcholine synthesis in canine tracheal smooth muscle. *FEBS Lett.* 267, 107–110.

Anonymous (1988). "Chemical Stockpile Disposal Program Final Programmatic Environmental Impact Statement." Appendix A, p. A3. Program manager for chemical demilitarization, Aberdeen Proving Ground, Maryland.

Anzueto, A., Berdine, G. G., Moore, G. T., Gleiser, C., Johnson, D., White, C. D., and Johanson, W. G., Jr. (1986). Pathophysiology of soman intoxication in primates. *Toxicol. Appl. Pharmacol.* **86**, 56–68.

Argyll-Robertson, D. (1863). On the Calabar bean as a new agent in ophthalmic practice. *Edinburgh Med. J.* **8**, 815–820.

Bellet, S. (1963). "Clinical Disorders of the Heart Beat." Lea and Febiger, Philadelphia, Pennsylvania.

Blennow, G., Brierley, J. B., Meldrum, B. S., and Siesjo, B. K. (1978). Epileptic brain damage. *Brain* **101**, 687–700.

Blick, D. W., Murphy, M. R., Fanton, J. W., Kerenyi, S. Z., Miller, S. A., and Hartgraves, S. L. (1989). Incapacitation and performance recovery after high-dose soman: Effects of diazepam. *Proceedings of the* 1989 *Medical Defense Bioscience Review*. AD B139550. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Bowers, M. B., Jr., Goodman, E., and Sim, V. M. (1964). Some behavioral changes in man following anticholinesterase administration. *J. Neurol. Ment. Dis.* **138**, 383–389.

Brown, E. C., Jr. (1948). "Effects of G-Agents on Man: Clinical Observations." Medical Division Report 158. Edgewood Arsenal, Maryland.

Calesnick, B., Christensen, J., and Richter, M. (1967). Human toxicity of various oximes. *Arch. Environ. Health* **15**, 599–608.

Craig, A. B., and Cornblath, M. (1953). "Further Clinical Observations in Workers Accidentally Exposed to G-Agents." Medical Laboratory Research Report 234. AD025225. Edgewood Arsenal, Maryland.

Craig, A. B., and Freeman, G. (1953). "Clinical Observations in Workers Accidentally Exposed to "G"-Agents." AD003393. Medical Laboratory Research Report 154. Edgewood Arsenal, Maryland.

Craig, A. B., and Woodson, G. S. (1959). Observations on the effects of accidental exposure to nerve gas. I. Clinical observations and cholinesterase depression. *Am. J. Med. Sci.* **238**, 49–53.

Craig, F. N., Cummings, E. G., and Sim, V. M. (1977). Environmental temperature and the percutaneous absorption of a cholinesterase inhibitor, VX. *J. Invest. Dermatol.* **68**, 357–361.

Davies, D. P., Green, A. C., and Willey, G. L. (1958). Hydroxyiminomethyl-*N*-methyl-pyridinium methane sulfonate and atropine in the treatment of severe organophosphate poisoning. *Br. J. Pharmacol.* **14**, 5–8.

Davis, W. (1985). "The Serpent and the Rainbow." Warner Books, New York.

DeCandole, C. A., Douglas, W. W., Evans, C. L., Holmes, R., Spencer, K. E. V., Torrance, R. W., and Wilson, K. M. (1953). The failure of respiration in death by anticholinesterase poisoning. *Br. J. Pharmacol.* **8**, 466–475.

deJong, L. P. A., and Wolring, G. Z. (1978). Effect of 1-(AR)alkyl-2-hydroxyiminomethylpyridinium salts on reactivation and aging of acetylcholinesterase inhibited by ethyl dimethylphosphoramidocyanidate (tabun). *Biochem. Pharmacol.* **27**, 2229–2235.

D'Mello, G. D., and Sidell, F. R. (1991). A model for carbamate and organophosphate-induced emesis in humans. *Neurosci. Biobehav. Rev.* **5**, 179–184.

Duffy, F. H., Burchfiel, J. L., Bartels, P. H., Gaon, M., and Sim, V. M. (1979). Long-term effects of an organophosphate upon the human electroencephalogram. *Toxicol. Appl. Pharmacol.* **47**, 161–176.

Edson, E. F. (1964). No-effect levels of three organophosphates in the rat, pig, and man. *Food Cosmet. Toxicol.* **2**, 311–316.

Fraser, T. R. (1863). On the characters, actions, and therapeutic use of the ordeal bean of calabar. *Edinburgh Med. J.* **9**, 124–132.

Freeman, G., Ludemann, H., Cornblath, M., Gold, A., Filbert, M., Udall, J., and Cugell, D. (1954). "Cardiovascular and Respiratory Effects of Acute Parathion Poisoning in Dogs with Particular Regard to Ventricular Fibrillation." Medical Laboratory Research Report 303. AD042287. Edgewood Arsenal, Maryland.

Ganendran, A. (1974). Organophosphate insecticide poisoning and its management. *Anaesth. Intensive Care.* 4, 361–368.

Grob, D. (1956). The manifestations and treatment of poisoning due to nerve gas and other organic phosphate anticholinesterase compounds. *Arch. Intern. Med.* 98, 221–239.

Grob, D., and Harvey, J. C. (1958). Effects in man of the anticholinesterase compound sarin (isopropyl methyl phosphonofluoridate). *J. Clin. Invest.* 37, 350–368.

Grob, D., Lilienthal, J. L., Jr., Harvey, A. M., and Jones, B. F. (1947). The administration of di-isopropyl fluorophosphate (DFP) to man. *Bull. Johns Hopkins Hosp.* 81, 217–244.

Harris, L. W., Heyl, W. C., Stitcher, D. L., and Broomfield, C. A. (1978). Effects of 1, 1-oxydimethylene-bis-(4-tert-butyl pyridinium chloride) (SAD-128) and decamethonium on reactivation of soman- and sarin-inhibited cholinesterase by oximes. *Biochem. Pharmacol.* 27, 757–761.

Harris, R., and Paxman, J. (1982). "A Higher Form of Killing." Hill and Wang, New York.

Harvey, J. C. (1952). "Clinical Observations on Volunteers Exposed to Concentrations of GB." Medical Laboratory Research Report 144. Edgewood Arsenal, Maryland.

Hassler, C. R., Moutvic, R. R., and Hamlin, R. L. (1987). Studies of the action of chemical agents on the heart. *Proceedings of the Sixth Medical Chemical Defense Bioscience Review.* AD B121516. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Hassler, C. R., Moutvic, R. R., Hobson, D. W., Lordo, R. A., Vinci, T., Dill, G. S., Joiner, R. L., and Hamlin, R. L. (1989). Long-term arrhythmia analysis of primates pretreated with pyridostigmine, challenged with soman, and treated with atropine and 2-PAMCl. *Proceedings of the 1989 Medical Chemical Defense Review.* AD B139550. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Hayes, G. R., Funckes, A. J., and Hartwell, W. J. (1964). Dermal exposure of human volunteers to parathion. *Arch. Environ. Health* 8, 829–833.

Hayes, W. (1982). "Pesticides Studied in Man." Williams & Wilkins, Baltimore, Maryland.

Hayward, I. J., Wall, H. G., Jaax, N. K., Wade, J. V., Marlow, D. D., and Nold, J. B. (1988). "Influence of Therapy with Anticonvulsant Compounds on the Effects of Acute Soman Intoxication in Rhesus Monkeys." USAMRICD TR-88-12. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Holmstedt, B. (1963). Structure-activity relationships of the organophosphorus anticholinesterase agents. *In* "Cholinesterases and Anticholinesterase Agents" (G. Koelle, ed.), pp. 429–431. Springer-Verlag, Berlin.

Johns, R. J. (1952). "The Effects of Low Concentrations of GB on the Human Eye." AD B9544842L. Medical Laboratory Research Report 100. Edgewood Arsenal, Maryland.

Johnson, R. P., Gold, A. J., and Freeman, G. (1958). Comparative lung-airway resistance and cardiovascular effects in dogs and monkeys following parathion and sarin intoxication. *Am. J. Physiol.* 102, 581–584.

Josselson, J., and Sidell, F. R. (1977). Dose-Response Effects of Intravenous Thiamine Hydrochloride on Pralidoxime Pharmacokinetics in Man." ADA037387. Edgewood Arsenal Technical Report EB-TR 76117. Edgewood Arsenal, Maryland.

Josselson, J., and Sidell, F. R. (1978). Effects of intravenous thiamine on pralidoxime kinetics. *Clin. Pharmacol. Ther.* 24, 95–100.

Kalow, W., and Genest, K. (1957). A method for the detection of atypical forms of human serum cholinesterase. Determination of dibucaine numbers. *Can. J. Biochem. Physiol.* 35, 339–346.

Karczmar, A. G. (1984). Acute and long-lasting central actions of organophosphorus agents. *Fundam. Appl. Toxicol.* **4**, S1–S17.

Kent, K. M., Epstein, S. E., Cooper, T., and Jacobowitz, D. M. (1974). Cholinergic innervation of the canine and human ventricular conducting system. *Circulation* **30**, 948–955.

Ketchum, J. S., Sidell, F. R., Crowell, E. B., Aghajanian, G. K., and Hayes, A. H. (1973). Atropine, scopolamine, and ditran: Comparative pharmacology and antagonists in man. *Psychopharmacology (Berlin)* **28**, 121–148.

Kiss, Z., and Fazekas, T. (1979). Arrhythmias in organophosphate poisonings. *Acta Cardiol. (Brux.)* **34**, 323–330.

Koelle, G. B. (1975). Anticholinesterase agents. *In* "The Pharmacological Basis of Therapeutics" (L. S. Goodman and A. Gilman, eds.), 5th Ed., p. 445. Macmillan, New York.

Koelle, G. B. (1981). Organophosphate poisoning—an overview. *Fundam. Appl. Toxicol.* **1**, 129–134.

Koplovitz, I., Harris, L. W., Anderson, D. R., Lennox, W. J., and Stewart, J. R. (1991). Reduction by pyridostigmine pretreatment of the efficacy of atropine and 2-PAM treatment of sarin and VX poisoning in rodents. *Fundam. Appl. Toxicol.* In press.

Kunkel, A. M., O'Leary, J. F., and Jones, A. H. (1973). "Atropine-Induced Ventricular Fibrillation during Cyanosis Caused by Organophosphorus Poisoning." AD758441: Edgewood Arsenal Technical Report 4711. Edgewood Arsenal, Maryland.

LeBlanc, F. N., Benson, B. E., and Gilg, A. D. (1986). A severe organophosphate poisoning requiring the use of an atropine drip. *Clin. Toxicol.* **24**, 69–76.

Lemercier, G., Carpentier, P., Sentenac-Roumanou, H., and Morelis, P. (1983). Histological and histochemical changes in the central nervous system of the rat poisoned by an irreversible anticholinesterase organophosphorus compound. *Acta Neuropathol. (Berlin)* **61**, 123–129.

Levin, H. S., and Rodnitzky, R. L. (1976). Behavioral effects of organophosphate pesticides in man. *Clin. Toxicol.* **9**, 391–405.

Lipp, J. A. (1973). Effect of benzodiazepine derivatives upon soman-induced seizure activity and convulsions in the monkey. *Arch. Int. Pharmacodyn. Ther.* **202**, 241–251.

Ludomirsky, A., Klein, H. O., Sarelli, P., Becker, B., Hoffman, S., Taitelman, U., Barzilai, J., Lang, R., David, D., DeSegni, E., and Kaplinsky, E. (1982). Q-T prolongation and polymorphous ("torsade de pointes") ventricular arrhythmias associated with organophosphorus insecticide poisoning. *Am. J. Cardiol.* **49**, 1654–1658.

Martin, L. J., Doebler, J. A., Shih, T., and Anthony, A. (1985). Protective effect of diazepam pretreatment on soman-induced brain lesion formation. *Brain Res.* **325**, 287–289.

McDonough, J. H., Jr., Jaax, N. K., Crowley, R. A., Mays, M. Z., and Modrow, H. E. (1989). Atropine and/or diazepam therapy protects against soman-induced neural and cardiac pathology. *Fundam. Appl. Toxicol.* **13**, 256–276.

McDonough, J. H., Jr., Smith, R. F., and Smith, C. D. (1986). Behavioral correlates of soman-induced neuropathology. Deficits in DRL acquisition. *Neurobehav. Toxicol. Teratol.* **8**, 179–187.

McDonough, J. H., Jr., McLeod, C. G., Jr., and Nipwoda, M. T. (1987). Direct microinjection of soman or VX into the amygdala produces repetitive limbic convulsions and neuropathology. *Brain Res.* **435**, 123–137.

McLeod, C. G., Jr., Singer, A. W., and Harrington, D. G. (1984). Acute neuropathology in soman-poisoned rats. *Neurotoxicology* **5**, 53–58.

Meldrum, B. S. (1983). Metabolic factors during prolonged seizures and their relation to nerve cell death. *In* "Advances in Neurology" (A. V. Delgado-Escueta, C. G. Wasterlain, D. M. Treiman, and R. J. Porter, eds.), pp. 261–275. Raven Press, New York.

Meldrum, B. S., and Brierley, J. B. (1973). Prolonged epileptic seizures in primates. *Arch. Neurol.* **28**, 10–17.

Modrow, H. W., and Jaax, N. K. (1987). Effect of soman exposure on the acquisition of an operant alternation task. *Pharmacol. Biochem. Behav.* **32**, 49–53.

Moylan-Jones, R. J., and Thomas, D. P. (1973). Cyclopentolate in treatment of sarin miosis. *Br. J. Pharmacol.* **48**, 309–313.

Namba, T., Nolte, C. T., Jackrel, J., and Grob, D. (1971). Poisoning due to organophosphate insecticides. *Am. J. Med.* **50**, 475–492.

Nevander, G., Ingvar, M., Auer, R., and Siesjo, B. K. (1985). Status epilepticus in well-oxygenated rats causes neuronal necrosis. *Ann. Neurol.* **18**, 281–290.

Oberst, F. W., Ross, R. S., Christensen, M. K., Crook, J. W., Cresthull, P., and Umland, B. S. (1956). Resuscitation of dogs poisoned by inhalation of the nerve gas GB. *Milit. Med.* **119**, 377–386.

O'Leary, J. F., Kunkel, A. M., and Jones, A. H. (1961) Efficacy and limitations of oxime-atropine treatment of organophosphorus anticholinesterase poisoning. *J. Pharm. Exp. Ther.* **132**, 50–57.

Orlowski, J. P., Erenberg, G., Lueders, H., and Cruse, R. P. (1984). Hypothermia and barbiturate coma for refractory status epilepticus. *Crit. Care Med.* **12**, 367–372.

Petras, J. M. (1981). Soman neurotoxicity. *Fundam. Appl. Toxicol.* **1**, 242.

Petras, J. M. (1984). Brain pathology induced by organophosphate poisoning with the nerve agent soman. *Proceedings of Fourth Annual Chemical Defense Bioscience Review*. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Raffaele, K., Hughey, D., Wenk, G., Olton, D., Modrow, H., and McDonough, J. (1987). Long-term behavioral changes in rats following organophosphonate exposure. *Pharmacol. Biochem. Behav.* **27**, 407–412.

Rengstorff, R. H. (1985). Accidental exposure to sarin. *Arch. Toxicol.* **56**, 201–203.

Rickett, D. L., Glenn, J. F., and Beers, E. T. (1986). Central respiratory effects versus neuromuscular actions of nerve agents. *Neurotoxicology* **7**, 225–236.

Rider, J. A., Mueller, H. C., Puletti, E. J., and Swader, J. I. (1969). Toxicity of parathion, systox, octamethyl pyrophosphoramide, and methyl parathion in man. *Toxicol. Appl. Toxicol.* **14**, 603–611.

Rider, J. A., Puletti, E. J., and Swader, J. I. (1975). The minimal oral toxicity level for mevinphos in man. *Tox. Appl. Pharmacol.* **32**, 97–100.

Robertson, G. S. (1967). Serum protein and cholinesterase changes in association with contraceptive pills. *Lancet* **i**, 232–235.

Robineau, P., and Guittin, P. (1987). Effects of an organophosphorus compound on cardiac rhythm and haemodynamics in anaesthetized and conscious beagle dogs. *Toxicol. Lett.* **37**, 95–102.

Robinson, J. P. (1971). "The Problem of Chemical and Biological Warfare. Volume I. The Rise of CB Weapons." Stockholm International Peace Research Institute. Humanities Press, New York.

Robinson, S., Buckingham, R. E., Pearcy, M., Smith, J. H., Daly, W., Maletich, R., Miller, D., and Robinson, D. (1953). 1952 Field Test of Atropine. *In* "The Physiological Effects of Atropine and Potential Atropine Substitutes" (S. Robinson, ed.), pp. 83–98. Medical Laboratory Contract Report 15, AD 019988. Aberdeen Proving Ground, Maryland.

Rubin, L. S., and Goldberg, M. N. (1958). Effects of tertiary and quaternary atropine salts on absolute scotopic threshold changes produced by an anticholinesterase (sarin). *J. Appl. Physiol.* **12**, 305–310.

Rubin, L. S., Krop, S., and Goldberg, M. N. (1957). Effect of sarin on dark adaptation in man. *J. Appl. Physiol.* **11**, 445–449.

Rump, S., Grudzinska, E., and Edelwein, Z. (1972). Effects of diazepam on abnormalities of bioelectrical activity of the rabbit's brain due to fluostigmine. *Act. Nerv. Super. (Prague)* **14**, 176–177.

Rump, S., Grundzinska, E., and Edelwein, Z. (1973). Effects of diazepam on epileptiform patterns of bioelectrical activity of the rabbit brain induced by fluostigmine. *Neuropharmacology* **12**, 815–819.

Seed, J. C. (1952). "An Accident Involving Vapor Exposure to Nerve Gas." Medical Laboratory Research Report 146. AD 000186. Edgewood Arsenal, Maryland.

Sidell, F. R. (1974). Soman and sarin: Clinical manifestations and treatment of accidental poisoning by organophosphates. *Clin. Toxicol.* **7**, 1–17.

Sidell, F. R., and Groff, W. A. (1971). Intramuscular and intravenous administration of small doses of 2-pyridinium aldoxime methochloride to man. *J. Pharm. Sci.* **60**, 1224–1228.

Sidell, F. R., and Groff, W. A. (1974). The reactivatibility of cholinesterase inhibited by VX and sarin in man. *Toxicol. Appl. Pharmacol.* **27**, 241–252.

Sidell, F. R., and Kaminskis, A. (1975a). Temporal intrapersonal physiological variability of cholinesterase activity in human plasma and erythrocytes. *Clin. Chem.* **21**, 1961–1963.

Sidell, F. R., and Kaminskis, A. (1975b). Influence of age, sex, and oral contraceptives on human blood cholinesterase activity. *Clin. Chem.* **21**, 1393–1395.

Sidell, F. R., Mershon, M. M., Savola, R. H., Schwartz, H. M., Wiles, J. S., and McShane, W. P. (1968). "Treatment of Percutaneous VX Intoxication in Rabbits under Conditions Simulating Self-Therapy in the Field." Edgewood Arsenal Technical Memorandum 114–22. AD841586. Edgewood Arsenal, Maryland.

Sidell, F. R., Groff, W. A., and Ellin, R. I. (1969). Blood levels of oxime and symptoms in humans after single and multiple doses of 2-PAMCl. *J. Pharm. Sci.* **58**, 1093–1098.

Sidell, F. R., Markis, J. E., Groff, W. A., and Kaminskis, A. (1974). Enhancement of drug absorption after administration by an automatic injector. *J. Pharmacokinet. Biopharm.* **2**, 197–210.

Sim, V. M. (1962). "Variability of Different Intact Human Skin Sites to the Penetration of VX." CRDL Report 3122. AD 271163. Edgewood Arsenal, Maryland.

Sim, V. M., and Stubbs, J. L. (1960). "VX Percutaneous Studies in Man." CRDL Report 3015. AD 318533. Edgewood Arsenal, Maryland.

Singer, A. W., Jaax, N. K., Graham, J. S., and McLeod, C. G., Jr. (1987). Cardiomyopathy in soman-and sarin-intoxicated rats. *Toxicol. Lett.* **36**, 243–249.

Slavikova, J., Vik, J., and Hlavickova, V. (1982). Acetylcholinesterase and butyrylcholinesterase activity in the atria of the heart of adult albino rats. *Physiol. bohemoslovaca* **31**, 407–414.

Smith, R. J. (1989). Army poison gas stockpile raises worries in Kentucky. *The Washington Post*. January 22, 1989. Washington, D.C.

Soderfeldt, B., Blennow, G., Kalimo, H., Olsson, Y., and Siesjo, B. K. (1983). Influence of systemic factors on experimental epileptic brain injury. *Acta Neuropathol.* **60**, 81–91.

Stewart, W. C., Madill, H. D., and Dyer, A. M. (1968). Night vision in the miotic eye. *Can. Med. Assoc. J.* **99**, 1145–1148.

Sundwall, A. (1961). Minimal concentrations of *N*-methyl-pyridinium-2-aldoxime methane sulfonate which reverse neuromuscular block. *Biochem. Pharmacol.* **8**, 413–417.

Swartz, R. D., and Sidell, F. R. (1974). Renal tubular secretion of pralidoxime in man. *Proc. Soc Exp. Biol. Med.* **146**, 419–424.

Swartz, R. D., and Sidell, F. R. (1973). Effects of heat and exercise on the elimination of pralidoxime in man. *Clin. Pharmacol. Ther.* **14**, 83–89.

Switzer, R. C., III, Campbell, S. K., Murphy, M. R., Kerenyi, S. Z., Miller, S. A., and Hartgraves, S. K. (1990). Soman-induced convulsions and brain damage as a function of chronic and acute exposure in rats and diazepam therapy in rhesus monkeys. *Proceedings of the Workshop on Convulsions and Related Brain Damage Induced by Organophosphorus Agents*. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Valero, A., and Golan, D. (1967). Accidental organophosphorus poisoning: The use of propranolol to counteract vagolytic cardiac effects of atropine. *Israel J. Med. Sci.* 3, 582–584.

Wall, H. G. (1987). Brain lesions in rhesus monkeys after acute soman intoxication. *Proceedings of the Sixth Medical Chemical Defense Bioscience Review.* AD B121516. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.

Whittaker, M., Charlier, A. R., and Ramaswamy, S. (1971). Changes in plasma cholinesterase isoenzyme due to oral contraceptives. *J. Reprod. Fertil.* 26, 373–375.

Willems, J., Vermeire, P., and Rolly, G. (1971). Some observations on severe human poisonings with organophosphate pesticides. *Arch. Toxicol.* 28, 182–191.

Wills, J. H., McNamara, B. P., and Fine, E. A. (1950). Ventricular fibrillation in delayed treatment of TEPP poisoning. *Fed. Proc.* 9, 136.

Wills, J. H., and DeArmon, I. A. (1954). "A Statistical Study of the Adamek Report." Medical Laboratory Special Report 54. AD045106. Edgewood Arsenal, Maryland.

Wilson, I. B. (1951). Acetylcholinesterase. XI. Reversibility of tetraethylpyrophosphate inhibition. *J. Biol. Chem.* 190, 111–117.

Wilson, I. B., and Ginsburg, S. (1955). A powerful reactivator of alkyl phosphate-inhibited acetylcholinesterase. *Biochim. Biophys. Acta* 18, 168–170.

Wright, P. G. (1954). An analysis of the central and peripheral components of respiratory failure produced by anticholinesterase poisoning in the rabbit. *J. Physiol.* 126, 52–70.

SUMMARY

Problem.

Recently, Persian Gulf War veteran research has been a major focus of federal and public attention. Due to the increasing interest and the need to continue to compile relevant references for the Gulf War investigators, we felt it was necessary to maintain a master bibliography for Persian Gulf War epidemiological research.

Objective.

The primary objective was to compile a master bibliography of the Persian Gulf War and related topics of research for federal investigators.

Approach.

This document was framed around a bibliography of the Persian Gulf War and associated topics, prepared by Jacqueline Van de Kamp, M.L.S., Specialized Information Services, National Library of Medicine, and John H. Ferguson, M.D., Office of Medical Applications of Research, National Institutes of Health. Their task was undertaken largely in preparation for the NIH Assessment Workshop on "The Persian Gulf Experience and Health," held in Bethesda, MD, April 27-29, 1994. Containing 594 citations, their bibliography became part of the National Library of Medicine's Current Bibliographies in Medicine (94-3).

We downloaded the Van de Kamp-Ferguson bibliography from the NIH's World Wide Webb site, and added to their work, revising the reference categories to reflect the changing trends in Persian Gulf research. While continuing to monitor for new references, we reviewed select references from the Van de Kamp-Ferguson bibliography, and additional references supplied by our colleagues. These manuscripts and their bibliographies led us to additional pertinent references, which we continue to add.

Results. We currently have 1,779 references, and we plan to continue expanding it, as more literature regarding the Persian Gulf War is published.

Conclusions. In addition to publication as a Navy Technical Document, copies of this work may be obtained from the Defense Technical Information Center's Gulflink on the World Wide Webb: http://www.dtic.dla.mil/gulflink. We will continue to update this bibliography on the Gulflink.

We appreciate suggestions for further additions, corrections, or improvements to this bibliography.

Authors **Internet mail address:**
Colleen McDonough Cornelison CORNELISON@VAX309.NHRC.NAVY.MIL
Gregory C. Gray, M.D., M.P.H. GRAY@VAX309.NHRC.NAVY.MIL

Doll R, Peto R. *The Causes of Cancer. Quantitative Estimates of Avoidable Risks of Cancer in the United States Today.* New York, Ny: Oxford University Press; 1981.

EPA (U.S. Environmental Protection Agency). Guidelines for carcinogen risk assessment. *Fed Regist.* 1986;51(185):33992-34003.

Kelly SJ, Guidotti TL. Phenoxyacetic acid herbicides and chlorophenols and the etiology of lymphoma and soft-tissue neoplasms. *Public Health Rev.* 1989-90;17(1):1-37.

Lilienfeld AM. Some aspects of cancer epidemiology. *Biometrics (Suppl). 1982;38:155-160.*

Mathews J. Panel urges further research on Gulf War illness, possible increased cancer risks in veterans [news]. *J Natl Cancer Inst.* 1994;86(11):820-822.

Settimi L, Boffetta P, Comba P, Terracini B. Epidemiologic study for the evaluation of the cancerogenic risk associated with pesticides. *Med Lav.* 1990;81(6):494-498.

CHEMICAL WARFARE

Aasted A, Darre E, Wulf HC. Mustard gas: clinical, toxicological, and mutagenic aspects based on modern experience. *Ann Plast Surg.* 1987;19(4):330-333.

Altenkirch H, Stoltenburg-Didinger G, Koeppel C. The neurotoxicological aspects of the toxic oil syndrome (TOS) in Spain. *Toxicology.* 1988;49(1):25-34.

Amitai Y, Almog S, Singer R, Hammer R, Bentur Y, Danon YL. Atropine poisoning in children during the Persian Gulf crisis: a national survey in Israel. *JAMA.* 1992;268(5):630-632.

Andreassi L. Chemical warfare and the skin. *Int J Dermatol.* 1991;30(4):252-253.

Anzueto A, de Lemos RA, Seidenfeld J, et al. Acute inhalation toxicity of soman and sarin in baboons. *Fundam Appl Toxicol.* 1990;14:676-687.

Balali M. Clinical and laboratory findings in Iranian fighters with chemical gas poisoning. *Arch Belg.* 1984(suppl):254-259.

Barnaby F. Iran-Iraq war: the use of chemical weapons against the Kurds. *Ambio.* 1988;17(6):407-408.

Barranco VP. Mustard gas and the dermatologist. *Int J Dermatol.* 1991;30(10):684-686.

Black RM, Clark RJ, Read RW, Reid MTJ. Application of gas chromatography-mass spectrometry and gas chromatography-tandem mass spectrometry to the analysis of chemical warfare samples, found to contain residues of the nerve agent sarin, sulphur mustard and their degradation products. *J Chromatogr A.* 1994;662:301-321.

Black RM, Read RW. Methods for the analysis of thiodiglycol sulphoxide, a metabolite of sulphur mustard, in urine using gas chromatography-mass spectrometry. *J Chromatogr.* 1991;558(2):393-404.

Blodi FC. Mustard gas keratopathy. *Int Ophthalmol Clin.* 1971;11(3):1-13.

Bondy SC. Especial considerations for neurotoxicological research. *Crit Rev Toxicol.* 1985;14(4):381-402.

Borak J, Sidell FR. Agents of chemical warfare: sulfur mustard. *Ann Emerg Med.* 1992;21(3):303-308.

Cancio LC. Chemical casualty decontamination by medical platoons in the 82nd Airborne Division. *Mil Med.* 1993;158(1):1-5.

Chan P. Recommended treatment for mustard casualties. In: *Medical Management of Chemical Casualties.* U.S. Army Medical Research Institute of Chemical Defense. 1988:6.28-6.32.

Charles D, Nowak R. The poisonous power of chemical warfare. *New Scientist.* 1990;Aug 25:22-24.

Clarkson TW. Metal toxicity in the central nervous system. *Environ Health Perspect.* 1987;75:59-64.

Crowell JA, Parker RM, Bucci TJ, Dacre JC. Neuropathy target esterase in hens after sarin and soman. *J Biochem Toxicol.* 1989;4:15-20.

Das Gupta S, Bass KN, Warnick JE. Interaction of reversible and irreversible cholinesterase inhibitors on the monosynaptic reflex in neonatal rats. *Toxicol Appl Pharmacol.* 1989;99:28-36.

ANTHRAX

Aleksandrov NI, Gefen NY, Garin NS, et al. Experiment of mass aerogenic vaccination of humans against anthrax. *Voyenno-Medilsinskii Zhural.* 1959;8:27-32.

Brachman PS, Gold H, Plotkin SA, Fekety FR, Werrin M, Ingraham NR. Field evaluation of a human anthrax vaccine. *Am J Public Health.* 1962;52:632-645.

Dickey C. His secret weapon. Iraq: Saddam had a big germ-warfare arsenal. *Newsweek.* September 4, 1995:34.

Friedlander AM, Welkos SL, Pitt MLM, et al. Postexposure prophylaxis against experimental inhalation anthrax. *J Infect Dis.* 1993;167:1239-1242.

Hambleton P, Carman JA, Melling J. Anthrax: the disease in relation to vaccines. *Vaccine.* 1984;2:125-132.

Howe EG. Medicine and war [letter]. *N Engl J Med.* 1992;327(15):1098.

Ivins BE, Welkos SL. Recent advances in the development of an improved, human anthrax vaccine. *Eur J Epidemiol.* 1988;4(1):12-19.

Ivins BE, Welkos SL, Knudson GB, et al. Transposon Tn916 mutagenesis in *Bacillus anthracis*. *Infect Immun.* 1988;56:176-181.

Ivins BE, Welkos SL, Knudson GB, et al. Immunization against anthrax with aromatic compound-dependent (Aro-) mutants of *Bacillus anthracis* and with recombinant strains of *Bacillus subtilis* that produce anthrax protective antigen. *Infect Immun.* 1990;58:303-308.

Kim WS, Chung SI, Choi CS, Yang YT. Association of pathogenicity of Genus Bacillus with the induction of superoxide dismutase in *Bacillus anthracis* and related Bacillus species. *Chung Ang J Med.* 1991;16(2):183-190.

Knudson GB. Treatment of anthrax in man: history and current concepts. *Mil Med.* 1986;151(2):71-77.

Knudson GB. Photoreactivation of ultraviolet-irradiated, plasmid-bearing, and plasmid-free strains of *Bacillus anthracis*. *Appl Environ Microbiol.* 1986;52:444-449.

Little SF, Knudson GB. Comparative efficacy of *Bacillus anthracis* live spore vaccine and protective antigen vaccine against anthrax in the guinea pig. *Infect Immun.* 1986;52:509-512.

Marshall E. U.S. bio-defenses faulted by GAO [news]. *Science.* 1991;251(4993):514.

McKee KT Jr, Berezuk GP, Balady MA. Medicine and war [letter]. *N Engl J Med.* 1992;327(15):1096-1097.

Metzger JF, Lewis GE Jr. Human-derived immune globulins for the treatment of botulism. *Rev Infect Dis.* 1979;1(4):689-692.

Nettleman MD. Biological warfare and infection control. *Infect Control Hosp Epidemiol.* 1991;12(6):368-372.

Nightingale SL. Medicine and war [letter]. *N Engl J Med.* 1992;327(15):1097-1098.

Titball RW, Turnbull PC, Hutson RA. The monitoring and detection of *Bacillus anthracis* in the environment. *Soc Appl Bacteriol Symp Ser.* 1991;20(suppl):9S-18S.

Turell MJ, Knudson GB. Mechanical transmission of *Bacillus anthracis* by stable flies (*Stomoxys calcitrans*) and mosquitoes (*Aedes Aegypti* and *Aedes taeniorhynchus*). *Infect Immun.* 1987;55:1859-1861.

Turnbull PC. Anthrax vaccines: past, present and future. *Vaccine.* 1991;9(8):533-539.

CANCER

Anderson GC. Cancer risks. War is unhealthy, US finds [news]. *Nature.* 1990;344(6266):478.

Bond GG, Bodner KM, Cook RR. Phenoxy herbicides and cancer: insufficient epidemiologic evidence for a causal relationship. *Fundam Appl Toxicol.* 1989;12(1):172-188.

Caldwell GG. Twenty-two years of cancer cluster investigations at the Centers for Disease Control. *Am J Epidemiol.* 1990;132(suppl):S43-S47.

Chemical Warfare

Dienstbier Z. Late effects of use of nuclear and certain chemical weapons in man. *Med War*. 1985;1(1):25-30.

Duffy B. The guns of August. *US News World Rep*. 1990;Aug 20:18-26.

Dunn MA, Sidell FR. Progress in medical defense against nerve agents. *JAMA*. 1989;262(5):649-652.

Gazzard MF, Thomas DP. A comparative study of central visual field changes induced by sarin vapor and physostigmine eye drops. *Exp Eye Res*. 1975;20:15-21.

Goldman M, Dacre JC. Lewisite: its chemistry, toxicology, and biological effects. *Rev Environ Contam Toxicol*. 1989;110:75-115.

Goldstein BD, Fincher DR, Searle JR. Electrophysiological changes in the primary sensory neuron following subchronic soman and sarin: alterations in sensory receptor function. *Toxicol Appl Pharmacol*. 1987;91:55-64.

Gupta RC, Patterson GT, Dettbarn WD. Comparison of cholinergic and neuromuscular toxicity following acute exposure to sarin and VX in rat. *Fundam Appl Toxicol*. 1991;16:449-458.

Hajgar J. Biological monitoring of exposure to nerve agents. *Br J Ind Med*. 1992;49:648-653.

Hedges SJ, Cart P, Fasulo L. Baghdad's dirty secrets: new Iraqi disclosures may help explain what ails Gulf-War veterans. *U.S. News and World Report*.

Heyndrickx A, Heyndrickx B. Treatment of Iranian soldiers attacked by chemical and microbiological war gases. *Riv Tossicol Sper Clin*. 1989;19(1-3):3-6.

Heyndrickx A, De Puydt H, Cordonnier J. Comparative study of two different field tests for the detection of yperite in the atmosphere, applied on biological samples of gased soldiers. *Arch Belg*. 1984;(suppl):61-68.

Heyndrickx A, Sookvanichsilp N, Van Den Heede M. Detection of trichothecene mycotoxins (Yellow Rain) in blood, urine, and faeces of Iranian soldiers treated as victims of a gas attack. *Riv Tossicol Sper Clin*. 1989;19(1-3):7-11.

Hu H, Cook-Deegan R, Shukri A. The use of chemical weapons. Conducting an investigation using survey epidemiology. *JAMA*. 1989;262(5):640-643.

Hughes JN, Knight R, Brown RFR, Marrs TC. Effects of experimental sarin intoxication on the morphology of the mouse diaphragm: a light and electron microscopical study. *Int J Exp Pathol*. 1991;72:195-209.

Husain K, Vijayaraghavan R, Pant SC, Raza SK, Pandey KS. Delayed neurotoxic effect of sarin in mice after repeated inhalation exposure. *J Appl Toxicol*. 1993;13:143-145.

Institute of Medicine. *Veterans at Risk: The Health Effects of Mustard Gas and Lewisite*. Pechura CM, Rall DP, eds. Washington, DC: National Academy Press; 1993.

Jacob WH, Friedl KE. Drug delivery systems for chemical defense. *Army Research, Development, and Acquisition Bulletin*. 1990;Jan/Feb:14-16.

Jenkins RA, Buchanen MV, Merriweather R, Ilgner RH, Gayle TM, Watson AP. Movement of chemical warfare agent stimulants through porous media. *J Hazard Mater*. 1994;37(2):303-325.

Kadivar H, Adams SC. Treatment of chemical and biological warfare injuries: insights derived from the 1984 Iraqi attack on Majnoon Island. *Mil Med*. 1991;156(4):171-177.

Kawabuchi M, Cintra WM, Despande SS, Albuquerque EX. Morphological and electrophysiological study of distal motor nerve fiber degeneration and sprouting after irreversible cholinesterase inhibition. *Synapse*. 1991;8:218-228.

Klein AK, Nasve ML, Goldman M. The effects of in vitro exposure to the neurotoxin sarin (GB) and soman (GD) on unscheduled DNA synthesis by rat hepatocytes. *Toxicol Lett*. 1987;38:239-249.

Kodavanti PRS, Mundy WR, Tilson HA, Harry GJ. Effects of selected neuroactive chemicals on calcium transporting systems in rat cerebellum and on survival of cerebellar granule cells. *Fundam Appl Toxicol*. 1993;21:308-316.

Kolka MA, Stephenson LA, Bruttig SP, et al. Human thermoregulation after atropine and/or pralidoxime administration. *Aviat Space Environ Med*. 1987;58:545-549.

Koslow EE. Would you go to war wearing this equipment? *Armed Forces Journal International*. 1990;May:55-58.

Chemical Warfare

Kuncl RW, George EB. Toxic neuropathies and myopathies. *Curr Opin Neurol.* 1993;6(5):695-704.

Little PJ, Scimeca JA, Martin BR. Distribution of [3H] diisopropyl-fluorophosphate, [3H] soman, [3H] sarin and their metabolites in mice brain. *Drug Metab Disposition.* 1988;16:515-520.

Liu DD, Watanabe HK, Ho IK, Hoskins B. Acute effects of Soman, Sarin, and Tabun on cyclic nucleotide metabolism in rat striatum. *J Toxicol Environ Health.* 1986;19:23-32.

Liverman CT, Pechura CM, compilers. *Health Effects of Mustard Gas and Lewisite: Subject Bibliography.* Washington, DC: Institute of Medicine, Division of Health Promotion and Disease Prevention;1992.

Macilain C. Study proves Iraq used nerve gas [news]. *Nature.* 1993;363:3.

Malizia E, Smeriglio M, Milletti M, et al. Acute poisoning with chemical warfare agents: clinical observations on a group of patients and a follow-up after seven years. *Riv Tossicol Sper Clin.* 1991;21(2-3):73-103.

Maurissen JP. Psychophysical testing in human populations exposed to neurotoxicants. *Neurobehav Toxicol Teratol.* 1985;7(4):309-317.

Meisenberg BR, Melaragno AJ, Monroy RL. Granulocyte colony stimulating factor (G-CSF) for mustard-induced bone marrow suppression. *Mil Med.* 1993;158(7):470-474.

Mellor SG, Rice P, Cooper GJ. Vesicant burns. *Br J Plast Surg.* 1991;44(6):434-437.

Mohamed-Ali H. Late lesions due to poison gas in survivors of the Iraqi poison warfare against the Kurdish people. *Wien Med Wochenschr.* 1992;142(1):8-15. (Ger).

Momeni AZ, Enshaeih S, Meghdadi M, Amindjavaheri M. Skin manifestations of mustard gas. A clinical study of 535 patients exposed to mustard gas. *Arch Dermatol.* 1992;128(6):775-780.

Morita H, Yanagisawa N, Nakajima T, et al. Sarin poisoning in Matsumoto, Japan. *Lancet.* 1995;346:290-293.

Munro NB, Watson AP, Ambrose KR, Griffin GD. Treating exposure to chemical warfare agents: implications for health care providers and community emergency planning. *Environ Health Perspect.* 1990;89:205-215.

Murray VS, Volans GN. Management of injuries due to chemical weapons [editorial]. *BMJ.* 1991;302(6769):129-130.

Mustard gas. *IARC Monogr Eval Carcinog Risk Chem Man.* 1975;9:181-192.

Newman-Taylor AJ, Morris AJ. Experience with mustard gas casualties [letter]. *Lancet.* 1991;337(8735):242.

Orma PS, Middleton RK. Aerosolized atropine as an antidote to nerve gas. *Ann Pharmacother.* 1992;26(7-8):937-938.

Pal T, Griffin GD, Miller GH, Watson AP, Daugherty ML, Vo-Dinh T. Permeation measurements of chemical agent simulants through protective clothing materials. *J Hazard Mater.* 1993;33(1):123-141.

Pauser G, Aloy A, Carvana M, et al. Lethal intoxication by wargases on Iranian soldiers. Therapeutic interventions on survivors of mustard gas and mycotoxin immersion. *Arch Belg.* 1984(suppl):341-351.

Pechura CM. From the Institute of Medicine. The health effects of mustard gas and lewisite. *JAMA.* 1993;269(4):453.

Pour-Jafari H, Moushtaghi AA. Alterations of libido in gased Iranian men. *Vet Hum Toxicol.* 1992;34(6):547.

Prakash UB. Chemical warfare and bronchoscopy [editorial]. *Chest.* 1991;100(6):1486.

Prevention and treatment of nerve gas poisoning. *Med Lett Drugs Ther.* 1990;32:103-105.

Rall DP, Pechura CM. Effects on health of mustard gas [letter]. *Nature.* 1993;366(6454):398-399.

Reed F. Gas warfare: plenty to worry about. *Army Times.* 1990;Sep 10:70.

Rees J, Harper P, Ellis F, Mitchell D. Mustard gas casualties [letter]. *Lancet.* 1991;337(8738):430.

Rengstorff RH. Accidental exposure to sarin: vision effects. *Arch Toxicol.* 1985;56:201-203.

Chemical Warfare

Requena L, Requena C, Sanchez M, et al. Chemical warfare. Cutaneous lesions from mustard gas. *J Am Acad Dermatol.* 1988;19(3):529-536.

Sandelowsky I, Simon GA, Bel P, Barak R, Vincze A. N1-(2-hydroxyethylthioethyl)-4-methyl imidazole (4-met-1-imid-thiodiglycol) in plasma and urine: a novel metabolite following dermal exposure to sulphur mustard. *Arch Toxicol.* 1992;66(4):296-297.

Schaumburg HH, Spencer PS. Recognizing neurotoxic disease. *Neurology.* 1987;37(2):276-278.

Scicchitano JP. Pentagon says Iraqi chemical threat is real. *Army Times.* 1990;Aug 20:16.

Seppalainen AM. Solvents and peripheral neuropathy. *Prog Clin Biol Res.* 1986;220:247-253.

Sidell FR. Clinical considerations in nerve agent intoxication. In: Somani SM, ed. *Chemical Warfare Agents.* Academy Press, Inc;1992:155-194.

Sidell FR. Medical ascpects of nerve agent exposure. In: *Medical Management of Chemical Casualties.* U.S. Army Medical Research Institute of Chemical Defense; 1988:5.9-5.21.

Sidell FR, Borak J. Chemical warfare agents: II. Nerve agents. *Ann Emerg Med.* 1992;21(7):865-871.

Singer AW, Jaax NK, Graham JS, McLeod CG Jr. Cardiomyopathy in Soman and Sarin intoxicated rats. *Toxicol Lett.* 1987;36:243-249.

Smith KJ, Hurst CG, Moeller RB, Skelton HG, Sidell FR. Sulfur mustard: its continuing threat as a chemical warfare agent, the cutaneous lesions induced, progress in understanding its mechanism of action, its long-term health effects, and new developments for protection and therapy. *Journal of the American Academy of Dermatology.* 1995;32(5):765-776.

Smith WJ, Dunn MA. Medical defense against blistering chemical warfare agents. *Arch Dermatol.* 1991;127(8):1207-1213.

Sohrabpour H. Clinical manifestations of chemical agents on Iranian combatants during Iran-Iraq conflict. *Arch Belg.* 1984;(suppl):291-297.

Soldiers as experimental animals [editorial]. *Nature.* 1993;361(6412):479.

Taher AA. Cleft lip and palate in Tehran. *Cleft Palate Craniofac J.* 1992;29(1):15-16.

Thomas TL, Kang HK. Mortality and morbidity among Army Chemical Corps Vietnam veterans: a preliminary report. *Am J Ind Med.* 1990;18(6):665-674.

Tice J. Liquid logistics imperative in the desert. *Army Times.* 1990;Aug 20:22.

Trueman RW. *Permethrin: Assessment for the Induction of Unscheduled DNA Synthesis in Primary Rat Hepatocyte Cultures.* CTL/P/1888. Imperial Chemical Industries Ltd; 1988.

Turque B, Glick D, Barry J. The specter of Iraq's poison gas. *Newsweek.* 1990;Aug 20:26.

van den Bercken J. The action of allethrin on the peripheral nervous system of the frog. *Pestic Sci.* 1977;8:692-699.

Verity MA. Neurotoxins and environmental poisons. *Curr Opin Neurol Neurosurg* 1992;5(3):401-405.

Warnick JE, Deshpande SB, Yang QZ, Das Gupta S. Biphasic action of sarin on monosynaptic reflex in the neonatal rat spinal cord in vitro. *Arch Toxicol.* 1993;67:302-306.

Watson AP, Ambrose KR, Griffin GD, Leffingwell SS, Munro NB, Waters LC. Health effects of warfare agent exposure: implications for stockpile disposal. *Environ Prof.* 1989;11(4):335-353.

Watson AP, Jones TD, Adams JD. Relative potency estimates of acceptable residues and reentry intervals after nerve agent release. *Ecotoxicol Environ Safety.* 1992;23(3):328-342.

Watson AP, Jones TD, Griffin GD. Sulfur mustard as a carcinogen: application of relative potency analysis to the chemical warfare agents H, HD, and HT. *Regul Toxicol Pharmacol.* 1989;10(1):1-25.

Watson R. Battle ready. *Newsweek.* 1990;Aug 20:20-25.

Wils ER, Hulst AG, de Jong AL, Verweij A, Boter HL. Analysis of thiodiglycol in urine of victims of an alleged attack with mustard gas. *J Anal Toxicol.* 1985;9(6):254-257.

Wils ER, Hulst AG, van Laar J. Analysis of thiodiglycol in urine of victims of an alleged attack with mustard gas, Part II. *J Anal Toxicol.* 1988;12(1):15-19.

Wright R. Iraqis admit to broad, virulent germ war plan. *Los Angeles Times.* September 6, 1995.

Yanagisawa N, ed. Report for toxic gas attack in Matsumoto. *Matsumoto Local Medical Council.* Matsumoto, Japan: 1995.

CHRONIC FATIGUE SYNDROME

Abbey SE, Garfinkel PE. Chronic fatigue syndrome and depression: cause, effect, or covariate. *Rev Infect Dis.* 1991;13(suppl):S73-S83.

Anderson N. Chronic fatigue syndrome: distinguish between syndromes and study them separately. *BMJ.* 1994;308:1298.

Barofsky I, Legro MW. Definition and measurement of fatigue. *Rev Infect Dis.* 1991;13(suppl):S94-S97.

Baschetti R. Viral illness and chronic fatigue (syndrome) [letter]. *Lancet.* 1995;346:47.

Bass C, Benjamin S. Management of chronic somatisation. *Br J Psychiatry.* 1993;162:472-478.

Bates DW, Schmitt W, Buchwald D, et al. Prevalence of fatigue and chronic fatigue syndrome in a primary care practice. *Arch Intern Med.* 1993;153:2259-2265.

Behan PO, Bakheit AM. Clinical spectrum of postviral fatigue syndrome. *Br Med Bull.* 1991;47(4):793-808.

Behan P, Behan W. The postviral fatigue syndrome. *Crit Rev Neurobiol.* 1988;42:157-178.

Bell DS. Chronic fatigue syndrome. Recent advances in diagnosis and treatment. *Postgrad Med.* 1992;91(6):245-252.

Bergner M, Bobbitt RA, Carter WB, Gilson BS. The sickness impact profile: development and final revision of a health status measure. *Med Care.* 1981;XIX:787-805.

Bertolin JM, Bertolin V. Chronic fatigue syndrome - biologic and psychopathological investigations. *Med Clin (Barc).* 1993;101(2):67-75.

Blondel-Hill E, Shafran SD. Treatment of the chronic fatigue syndrome. A review and practical guide. *Drugs.* 1993;46(4):639-651.

Bond PA. A role for herpes simplex virus in the aetiology of chronic fatigue syndrome and related disorders. *Med Hypotheses.* 1993;40(5):301-308.

Bruce-Jones WDA, White PD, Thomas JM, Clare AW. The effect of social adversity on the fatigue syndrome, psychiatric disorders and physical recovery, following glandular fever. *Psychol Med.* 1994;24:651-659.

Buchwald D, Cheney PR, Peterson DL, et al. A chronic illness characterized by fatigue, neurologic and immunologic disorders, and active human herpesvirus type 6 infections. *Ann Intern Med.* 1992;116:103-113.

Buchwald D, Garrity D. Comparison of patients with Chronic Fatigue Syndrome, fibromyalgia, and multiple chemical sensitivities. *Arch Intern Med.* 1994;154:2049-2053.

Buchwald D, Garrity D, Pascualy R, et al. Chronic fatigue syndrome. *Toxicol Ind Health.* 1992;8(4):157-173.

Buchwald D, Komaroff AL. Review of laboratory findings for patients with chronic fatigue syndrome. *Rev Infect Dis.* 1991;13(suppl 1): 512-518.

Buchwald D, Umall P, Umall J, Kith P, Pearlman T, Komaroff A. Chronic fatigue and the chronic fatigue syndrome: prevalence in a Pacific Northwest health care system. *Ann Intern Med.* 1995;123(2):81-88.

Byrne E. The chronic fatigue syndrome: a reappraisal and unifying hypothesis. *Clin Exp Neurol.* 1991;28:128-138.

Caligiuri M, Murray C, Buchwald D, Levine H, Cheney P, Peterson D. Phenotypic and functional deficiency of natural killer cells in patients with chronic fatigue syndrome. *J Immunol.* 1987;139(10):3306-3313.

Chalder T, Berelowitz G, Pawlikowska T, et al. Development of a fatigue scale. *J Psychosom Res.* 1993;37:147-153.

Cohen S, Tyrrell DAJ, Smith AP. Negative life events, perceived stress, negative affect and susceptibility to the common cold. *J Pers Soc Psychol.* 1993;64:131-140.

Chronic Fatigue Syndrome

Cope H, David A, Mann A. "Maybe it's a virus": beliefs about viruses, symptom attributional style and psychological health. *J Psychosomatic Res.* 1994;38:89-98.

Cope H, David A, Pelosi A, Mann A. Predictors of chronic "postviral" fatigue. *Lancet.* 1994;344:864-868.

Croft P, Ridby A, Boswell R, Schollum J, Silman A. The prevalence of chronic widespread pain in the general population. *J Rheumatol.* 1993;20:710-713.

Daugherty SA, Henry BE, Peterson DL, et al. Chronic fatigue syndrome in northern Nevada. *Rev Infect Dis.* 1991;13(suppl 1):539-544.

David A. The postviral fatigue syndrome and psychiatry. *Br Med Bull.* 1991;47:966-988.

David A, Cope H, Pelosi A, Mann A. Viral illness and chronic fatigue (syndrome) [letter]. *Lancet.* 1995;346:47.

David A, Pelosi A, McDonald E, et al. Tired, weak or in need of rest: fatigue among general practice attenders. *BMJ.* 1990;301:1199-1202.

David A, Pelosi A, Wessely S. The chronic fatigue syndrome ("ME"): signs of a new approach. *Br J Hosp Med.* 1991;45:158-163.

David A, Wessely S. Chronic fatigue, ME, and ICD-10. *Lancet.* 1993;342:1247-1248.

David A, Wessely S, Pelosi A. Postviral fatigue syndrome: time for a new approach. *BMJ.* 1988;296:696-699.

Dodge JH, Kita MW. The chronic fatigue syndrome [letter]. *Ann Int Med.* 1995;123(1):75.

Evans AS. Chronic fatigue syndrome: thoughts on pathogenesis. *Rev Infect Dis.* 1991;13(suppl 1):556-559.

Fark AR. Infectious mononucleosis, Epstein-Barr virus, and chronic fatigue syndrome: a prospective case series. *J Fam Pract.* 1991;32(2):202-209.

Fukuda K, Straus SE, Hickie I, et al. The chronic fatigue syndrome: a comprehensive approach to its definition and study. *Ann Intern Med.* 1994;121:953-959.

Fukuda K, Strauss SE. The chronic fatigue syndrome [response]. *Ann Int Med.* 1995;123(1):76.

Galbraith DN, Naim C, Clements GB. Phylogenetic analysis of short enteroviral sequences from patients with chronic fatigue syndrome. *Journal of General Virology.* 1995;76:1701-1707.

Goldenberg DL. Fibromyalgia, chronic fatigue, and myofascial pain syndromes. *Curr Opin Rheumatol.* 1992;4(2):247-257.

Gow JW, Behan WMH, Clements GB, et al. Enteroviral RNA sequences detected by PCR in muscle of patients with postviral fatigue syndrome. *BMJ.* 1991;302:692-696.

Gow JW, Behan WMH, Simpson K, et al. Studies on enterovirus in patients with chronic fatigue syndrome. *Clin Infect Dis.* 1994;Jan 18 (suppl 1):126-129.

Gow JW, Simpson K, Schliephake A, et al. Search for retrovirus in chronic fatigue syndrome. *J Clin Pathol.* 1992;45:1058-1061.

Greenberg DB. Neurasthenia in the 1980s: chronic mononucleosis, chronic fatigue syndrome, and anxiety and depressive disorders. *Psychosomatics.* 1990;31(2):129-137.

Grufferman S. Issues and problems in the conduct of epidemiologic research on chronic fatigue syndrome. *Rev Infect Dis.* 1991;13(suppl):S60-S67.

Gunn JW, Connell DB, Randall B. Epidemiology of chronic fatigue syndrome: the Centers for Disease Control study. In: Buck G, Whelan J, eds. *Chronic Fatigue Syndrome.* New York: Wiley; 1993:83-101.

Hansen V, Jacobsen BK. Mental distress and social conditions and lifestyle in northern Norway. *BMJ.* 1989;299:85-88.

Harvey WT. A flight surgeon's personal view of an emerging illness. *Aviat Space Environ Med.* 1989;60(12):1199-1201.

Henderson DA. Reflections on epidemic neuromyasthenia (chronic fatigue syndrome). *Clin Infect Dis.* 1994;18(suppl):S3-S6.

Holmes GP. The chronic fatigue syndrome. In: Schlossberg D, ed. *Infectious Mononycleosis.* 2nd ed. New York: Springer-Verlag; 1989:172-93.

Holmes GP, Kaplan JE, Gunitz NM, et al. Chronic fatigue syndrome: a working case definition. *Ann Intern Med.* 1988;108:387-389.

Chronic Fatigue Syndrome

Holmes GP, Kaplan JE, Schonberger LB, et al. Definition of the chronic fatigue syndrome [letter]. *Ann Intern Med.* 1988;109:512.

Imboden J, Canter A, Cluff L. Convalescence from influenza: a study of the psychological and clinical determinants. *Arch Intern Med.* 1961;108:115-121.

Jamal GA, Miller RG. Neurophysiology of postviral fatigue syndrome. *Br Med Bull.* 1991;47(4):815-825.

James DG, Brook MG, Bannister BA. The chronic fatigue syndrome. *Postgrad Med J.* 1992;68(802):611-614.

Jones JF. Epstein-Barr virus and the chronic fatigue syndrome: a short review. *Microbiol Sci.* 1988;5(12):366-369.

Jones JF. Serologic and immunologic responses in chronic fatigue syndrome with emphasis on the Epstein-Barr virus. *Rev Infect Dis.* 1991;13(suppl 1):526-531.

Katon WJ, Buchwald DS, Simon GE, et al. Psychiatric illness in patients with chronic fatigue and rheumatoid arthritis. *J Gen Intern Med.* 1991;6:227-285.

Katon W, Russo J. Chronic fatigue syndrome criteria: a critique of the requirement for multiple physical complaints. *Arch Intern Med.* 1992;152:1604-1609.

Kleinmann A, Straus SE. *Chronic fatigue syndrome.* Ciba Foundation Symposium 173. Chichester: John Wiley & Sons; 1993.

Klonoff DC. Chronic fatigue syndrome. *Clin Infect Dis.* 1992;15:812-823.

Komaroff A, Buchwald D. Symptoms and signs in chronic fatigue syndrome. *Rev Infect Dis.* 1991;13(suppl):S8-S11.

Komaroff AL. Clinical presentation of chronic fatigue syndrome. *Ciba Found Symp.* 1993;173:43-54.

Krilov LR. Chronic fatigue syndrome. *Pediatric Annals.* 1995;24(6):290-292, 294.

Kroenke K. Chronic fatigue: frequency, causes, evaluation, and management. *Compr Ther.* 1989;15:3-7.

Kroenke K, Wood DR, Mangelsdorff AD, Meier NJ, Powell JB. Chronic fatigue in primary care: prevalence, patient characteristics, and outcome. *JAMA.* 1988;206:929-934.

Kruesi MJ, Dale J, Straus SE. Psychiatric diagnoses in patients who have chronic fatigue syndrome. *J Clin Psychiatry.* 1989;50:53-56.

Krupp LB, LaRocca NG, Muir-Nash J, Steinberg AD. The fatigue severity scale: application to patients with multiple sclerosis and systemic lupus erythematosis. *Arch Neurol.* 1989;46:1121-1123.

Krupp LB, Mendelson WB, Friedman R. An overview of chronic fatigue syndrome. *J Clin Psychiatry.* 1991;52:403-410.

Kutemeyer M. Chronic fatigue syndrome - new clinical diagnosis. *Nervenarzt.* 1991;62(1):64-66.

Landay AL, Jessop C, Lennette ET, Levy JA. Chronic fatigue syndrome: clinical condition associated with immune activation. *Lancet.* 1991;338:707-712.

Lane TJ, Matthews DA, Manu P. The low yield of physical examination and laboratory investigations of patients with chronic fatigue. *Am J Med Sci.* 1990;299:313-318.

Lapp CW, Cheney PR. The chronic fatigue syndrome [letter]. *Ann Int Med.* 1995;123(1):74-75.

Lawrie SM, Pelosi AJ. Chronic fatigue syndrome in the community: prevalence and associations. *Br J Psychiatry.* 1995;166:793-797.

Levine PH, Jacobson S, Pocinki AG, et al. Clinical, epidemiologic, and virologic studies in four clusters of the chronic fatigue syndrome. *Arch Intern Med.* 1992;152:1611-1616.

Lewis G, Wessely S. The epidemiology of fatigue: more questions than answers. *J Epidemiol Community Health.* 1992;46:92-97.

Lloyd AR, Hickie I, Boughton CR, Spencer O, Wakefield D. Prevalence of chronic fatigue syndrome in an Australian population. *Med J Aust.* 1990;153:522-528.

Macintyre A. Viral illness and chronic fatigue (syndrome) [letter]. *Lancet.* 1995;346:47.

Chronic Fatigue Syndrome

Mant D. Chronic fatigue syndrome [letter]. *Lancet.* 1994;344:834-835.

Manu P, Lane TJ, Matthews DA. Somatization disorder in patients with chronic fatigue. *Psychosomatics.* 1989;30:388-395.

Manu P, Matthews DA, Lane TJ. The mental health of patients with a chief complaint of chronic fatigue: a prospective evaluation and follow-up. *Arch Intern Med.* 1988;148:2213-2217.

Manu P, Matthews DA, Lane TJ. Panic disorder among patients with chronic fatigue. *South Med J.* 1991;84:451-456.

Matthews DA, Lane TJ, Manu P. Definition of the chronic fatigue syndrome [letter]. *Ann Intern Med.* 1988;109:511-512.

Mawle AC, Reyes M, Schmid DS. Is chronic fatigue syndrome an infectious disease? *Infect Agents Dis.* 1994;2:333-341.

McDonald E, David A, Pelosi A, et al. Chronic fatigue in general practice attenders. *Psychol Med.* 1993;23:987-988.

Miller NA, Carmichael HA, Calder BD, et al. Antibody to coxsackie B virus in diagnosing postviral fatigue syndrome. *BMJ.* 1991;302:140-143.

Moldofsky H. Fibromyalgia, sleep disorder and chronic fatigue syndrome. *Ciba Found Symp.* 1993;173:262-271.

Nixon PG. The grey area of effort syndrome and hyperventilation: from Thomas Lewis to today. *J R Coll Physicians Lond.* 1993;27(4):377-383.

Pawlikowska T, Chalder T, Hirsch SR, Wallace P, Wright DJ, Wessely SC. Population based study of fatigue and psychological distress. *BMJ.* 1994;308:763-766.

Peterson PK, Schenck CH, Sherman R. Chronic fatigue syndrome in Minnesota. *Minn Med.* 1991;74:21-26.

Piper BF, Lindsey AM, Dodd MJ, Perketich S, Paul SM, Weller S. The development of an instrument to measure the subjective dimension of fatigue. In: Funk SG, Tournquist PM, Campagne MT, Archer Gopp L, Wiese RA, eds. *Key Aspects of Comfort: Management of Pain, Fatigue and Nausea.* New York, Ny: Springer; 1989:199-208.

Powell R, Dolan R, Wessely S. Attributions and self-esteem in depression and chronic fatigue syndromes. *J Psychosom Res.* 1990;34:665-673.

Price RK, North CS, Wessely S, Frazer VJ. Estimating the prevelance of chronic fatigue syndrome and associated symptoms in the community. *Public Health Rep.* 1992;107:514-522.

Prince K. Chronic fatigue syndrome: keep an open mind. *BMJ.* 1994;308:1300.

Ramsay M. *Postviral Fatigue Syndrome: The Saga of Royal Free Disease.* London, Eng: Gower Medical; 1986.

Rest J. The chronic fatigue syndrome [letter]. *Ann Int Med.* 1995;123(1):75.

Ridsdale L, Evans A, Jerrett W, et al. Patients with fatigue in general practice: a prospective study. *BMJ.* 1993;307:103-106.

Robbins JM, Kirmayer LJ. Attributions of common somatic symptoms. *Psychol Med.* 1991;21:1029-1045.

Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of Mental Health Diagnostic Interview Schedule: its history, characteristics, and validity. *Arch Gen Psychiatry.* 1981;38:381-389.

Robins LN, Wing J, Wittchen HU, et al. The Composite International Interview: an epidemiologic instrument suitable for use in conjunction with different diagnostic systems and in different cultures. *Arch Gen Psychiatry.* 1988;45:1069-1077.

Rotheram EB Jr. The chronic fatigue syndrome [letter]. *Ann Int Med.* 1995;123(1):75.

Rutherford OM, White PD. Human quadriceps strength and fatiguability in patients with post viral fatigue. *J Neurol Neurosurg Psychiatry.* 1991;54:961-964.

Schluederberg A, Straus SE, Peterson P, et al. Chronic fatigue syndrome research: definition and medical outcome assessment. *Ann Intern Med.* 1992;117:325-331.

Schmidt P, Blanck RM. Gulf War Syndrome and CFS. *CFIDS Chron.* 1995;8:25-27.

Schwartz JE, Jandorf L, Krupp LB. The measurement of fatigue: a new instrument. *J Psychosom Res.* 1993;37:753-762.

Shafran SD. The chronic fatigue syndrome. *Am J Med.* 1991;90:730-739.

Sharpe MC, Archard LC, Banatvala JE, et al. A report - chronic fatigue syndrome: guidelines for research. *J R Soc Med.* 1991;84:118-121.

Sharpe M, Hawton K, Seagroatt V, Pasvol G. Follow-up of patients presenting with fatigue to an infectious disease clinic. *BMJ.* 1992;305:147-152.

Shave DW. A flight surgeon's personal view of an emerging illness [letter]. *Aviat Space Environ Med.* 1990;61(5):443-444.

Shepherd C. Viral illness and chronic fatigue (syndrome) [letter]. *Lancet.* 1995;346:47-48.

Spitzer RL, Williams JB, Gibbon M, First MB. The structured clinical interview for DSM-III-R (SCID). I: History, rationale, and description. *Arch Gen Psychiatry.* 1992;49:624-629.

Straus SE. Defining the chronic fatigue syndrome [editorial]. *Arch Intern Med.* 1992;152:1569-1570.

Straus S, Tosato G, Armstrong G, et al. Persisting illness and fatigue in adults with evidence of Epstein-Barr virus infection. *Ann Intern Med.* 1985;102:7-16.

Sumaya CV. Serologic and virologic epidemiology and Epstein-Barr virus: relevance to chronic fatigue syndrome. *Rev Infect Dis.* 1991;13(suppl 1): 519-525.

Swartz MN. The chronic fatigue syndrome - one entity or many? *N Engl J Med.* 1988;319:1726-1728.

Tannock C, Costa DC, Brostoff J. Chronic fatigue syndrome: preliminary report misrepresented. *BMJ.* 1994;308:1300.

Thomas PK. The chronic fatigue syndrome: what do we know? *BMJ.* 1993;306:1557-1558.

Vercoulen JH, Swanink CM, Fennis JF, Galama JM, van der Muer JW, Bleijenberg G. Dimensional assessment of chronic fatigue syndrome. *J Psychosom Res.* 1994;38:383-392.

Walker EA, Katon WJ, Jemelka RP. Psychiatric disorders and medical care utilization among people in the general population who report fatigue. *J Gen Intern Med.* 1993;8:436-440.

Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36): I. conceptual framework and item selection. *Med Care.* 1992;30:473-483.

Ware NC, Kleinman A. Culture and somatic experience: the social course of illness in neurasthenia and chronic fatigue syndrome. *Psychosom Med.* 1992;54(5):546-560.

Welch JC. Chronic fatigue syndrome and liquorice [letter]. *N Z Med J.* 1995;June 14:234-235.

Wessely S. Old wine in new bottles: neurasthenia and "ME." *Psychol Med.* 1990;20:35-53.

Wessely S. History of the postviral fatigue syndrome. *Br Med Bull.* 1991;47:919-941.

Wessely S, Chalder T, Hirsch S, Pawlikowska T, Wallace P, Wright DJM. Postinfectious fatigue: prospective cohort study in primary care. *Lancet.* 1995;345:1333-1338.

Wessely S, Powell R. Fatigue syndromes: a comparison of chronic postviral fatigue with neuromuscular and affective disorders. *J Neurol Neurosurg Psychiatry.* 1989;52:940-948.

Wilson A, Hickie I, Lloyd A, et al. Longitudinal study of outcome of chronic fatigue syndrome. *BMJ.* 1994;308:756-759.

Wilson A, Hickie I, Lloyd A, Wakefield D.v The treatment of chronic fatigue syndrome: science and speculation. *Am J Med.* 1994;96:544-550.

Wood GC, Bentall RP, Gopfert M, et al. A comparative psychiatric assessment of patients with chronic fatigue syndrome and neuromuscular disease. *Psychol Med.* 1991;21:619-628.

Wookey C. Viral illness and chronic fatigue (syndrome) [letter]. *Lancet.* 1995;346:48.

Zajdowicz TR. Chronic fatigue syndrome and military service [letter]. *Mil Med.* 1992;157(9):A3-A4.

FIBROMYALGIA

Bennett RM. Fibromyalgia and the facts. Sense or nonsense. *Rheum Dis Clin North Am.* 1993;19(1):45-59.

Gastrointestinal Disease

Ashkenazi S, Cleary KR, Pickering LK, et al. The association of Shiga toxin and other cytotoxins with the neurologic manifestations of shigellosis. *J Infect Dis*. 1990;161:961-965.

Asperilla MO, Smego RA Jr, Scott LK. Quinolone antibiotics in the treatment of *Salmonella* infections. *Rev Infect Dis*. 1990;12:873-889.

Avery ME, Snyder ID. Oral therapy for acute diarrhea: the underused simple solution. *N Engl J Med*. 1990;323:891-894.

Bassily S, Farid Z, Lehman JS Jr, Kent DC, Sanborn WR, Hathout SD. Treatment of chronic urinary *salmonella* carriers. *Trans R Soc Trop Med Hyg*. 1970;64:723-729.

Beers LM, Burke TL, Martin DB. Shigellosis occurring in newborn nursery staff. *Infect Control Hosp Epidemiol*. 1989;10:147-149.

Bennish ML, Salam MA, Haider R, et al. Therapy for shigellosis. II. Randomized double-blind comparison of ciprofloxacin and ampicillin. *J Infect Dis*. 1990;162:711-716.

Bratoeva MP, John JF. Dissemination of trimethoprim-resistant clones of *Shigella sonnei* in Bulgaria. *J Infect Dis*. 1989;159:648-653.

Chalapati VR, Theodore GM, Melnick JL. Human viruses in sediments, sludges and soils. *Bull WHO*. 1986;64:1-14.

Chugh TD. Transferable resistance to trimethoprim in enteric pathogens isolated in Kuwait. *J Hyg (Lond)*. 1985;95:391-395.

Chugh TD, Suheir A. Drug resistance among *Salmonellae* in Kuwait. *Trop Geogr Med* 1983;35:37-41.

Cohen D, Green M, Block C, et al. Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet* 1991;337:993-997.

Csaki F, Endredi I. Pollution by nitrates of the subsurface waters in Hungary. In: Duijvenbooden WV, Glasbergen P, Lelyveld HV, eds. *Quality of Groundwater*. The Netherlands: Elsevier Scientific Publishing Co.; 1981:89-94.

Damjanovic V, Furtado M, Patmore M. Antibiotic sensitivity of enteropathogenic bacteria isolated from patients in a Sharjah hospital. *J Hyg (Lond)*. 1984; 92:205-208.

Daniell FD, Crafton LD, Waiz SE, Bolton HT. Field preventive medicine and epidemiologic surveillance: the Beirut, Lebanon experience, 1982. *Mil Med*. 1985;150:171-176.

Davis H, Taylor JP, Perdue JN, et al. A shigellosis outbreak traced to commercially distributed shredded lettuce. *Am J Epidemiol*. 1988;128:1312.

Dean AG, Ching Y-C, Williams RG, Harden LB. Test for *Escherichia coli* enterotoxin using infant mice: application in a study of diarrhea in children in Honolulu. *J Infect Dis*. 1972;125:407-411.

DeMaio J, Hall K, Bailey L, Boyd R. A major outbreak of foodborne gastroenteritis among Air Force personnel during Operation Desert Storm. *Mil Med*. 1993;158:161-164.

Denbert ML, Sorensen AL, Perez JD, et al. Shipboard investigation of intestinal salmonellosis. *Navy Med*. 1988;79:26-28.

Drugs for parasitic infections. *Med Lett Drugs Ther*. 1990;32:23-30.

DuPont HL. Shigella species (bacillary dysentery). In: Mandell GL, Douglas RG, Bennett JE, eds. *Principles and Practice of Infectious Disease. 3rd ed*. New York, Ny: Churchill Livingstone; 1990:1716-1722.

DuPont HL, Ericsson CD, Robinson A, et al. Current problems in antimicrobial therapy for bacterial enteric infection. *Am J Med*. 1987;82:324-328.

DuPont HL, Hornick RB. Adverse effect of Lomotil therapy in shigellosis. *JAMA*. 1973;82:1073-1081.

DuPont HL, Levine MM. Hornick RB, Formal SB. Inoculum size in shigellosis and implications for expected mode of transmission. *J Infect Dis*. 1989;159:1126-1128.

Duijvenbooden WV, Loch JP. Nitrate in the Netherlands - a serious threat to groundwater. *Aqua*. 1983;2:59-63.

Echeverria P, Blacklow NR, Zipkin C, et al. Etiology of gastroenteritis among Americans living in the Philippines. *Am J Epidemiol*. 1979;109:493-501.

Echeverria P, Hodge FA, Blacklow NR, et al. Travelers' diarrhea among United States Marines in South Korea. *Am J Epidemiol*. 1978;108:68-73.

Gastrointestinal Disease

Block SR. Fibromyalgia and the rheumatisms. Common sense and sensibility. *Rheum Dis Clin North Am.* 1993;19(1):61-78.

Buchwald D, Garrity D. Comparison of patients with Chronic Fatigue Syndrome, fibromyalgia, and multiple chemical sensitivities. *Arch Intern Med.* 1994;154:2049-2053.

Cohen ML, Quintner JL. Fibromyalgia syndrome, a problem of tautology. *Lancet.* 1993;342(8876):906-909.

Culclasure TF, Enzenauer RJ, West SG. Post-traumatic stress disorder presenting as fibromyalgia. *Am J Med.* 1993;94(5):548-549.

Duna GF, Wilke WS. Diagnosis, etiology, and therapy of fibromyalgia. *Compr Ther.* 1993;19(2):60-63.

Goldenberg DL. Fibromyalgia syndrome. An emerging but controversial condition. *JAMA.* 1987;257(20):2782-2787.

Goldenberg DL. Fibromyalgia and its relationship to chronic fatigue syndrome, viral infection and immune abnormalities. *J Rheumatol.* 1989;16(suppl):91-93.

Goldenberg DL. Fibromyalgia, chronic fatigue, and myofascial pain syndromes. *Curr Opin Rheumatol.* 1992;4(2):247-257.

Hudson JI, Goldenberg DL, Pope HG, Keck PE, Schiesinger L. Comorbidity of fibromyalgia with medical and psychiatric disorders. *AM J Med.* 1992;92:363-367.

Kersley G. Primary fibromyalgia syndrome (5). *Br J Rheumatol.* 1993;32(7):646.

Moldofsky H. Fibromyalgia, sleep disorder and chronic fatigue syndrome. *Ciba Found Symp.* 1993;173:262-271.

Powers R. Fibromyalgia: an age-old malady begging for respect. *J Gen Intern Med.* 1993;8(2):93-105.

Rothschild BM. Fibromyalgia: an explanation for the aches and pains of the nineties. *Compr Ther.* 1991;17(6):9-14.

Visuri T, Lindholm H, Lindqvist A, Dahlstrom S, Viljanen A. Cardiovascular functional disorder in primary fibromyalgia: a noninvasive study in 17 young men. *Arthritis Care Res.* 1992;5(4):210-215.

Young MB, Mass AT, Alged JC. A controlled study of primary fibromyalgia syndrome: clinical features and association with other functional syndromes. *J Rheumatol.* 1989;16(suppl 19):62-71.

GASTROINTESTINAL DISEASE

Abdel-Hafez MM, el-Kady N, Bolbol AS, Baknina MH. Prevalence of intestinal parasitic infections in Riyadh district, Saudi Arabia. *Ann Trop Med Parasitol.* 1986;80:631-634.

Abdul-Fattah AF, Husseiny AA, Sabri ZA. Desalting in Saudi Arabia - demand production, management, associated power generation and future plans. *Desalination.* 1978;25:9-44.

Abou Gamra EM, El-Rahman Moussa MA, Fathi OM, Soliman H, Badawi N. Endemicity of giardiasis in Saudi Arabia. *J Egypt Soc Parasitol.* 1983;13:227-229.

Adkins H, Merrell B, O'Rourke T, et al. Travelers' diarrhea among U.S. Navy and Marine Corps personnel during a Western Pacific deployment. *Mil Med.* 1990;155:111-116.

Ahmed MM, Bolbol AH. The intestinal parasitic infections among children in Riyadh, Saudi Arabia. *J Egypt Soc Parasitol.* 1989;19:583-588.

Alaa-el-Din MN, Madany IM, Al-Tayaran A, Al-Jubair AH, Gomaa A. Quality of water from some wells in Saudi Arabia. *Wat Air Soil Pollut.* 1992;66:135-143.

Alaa-el-Din MN, Madany IM, Al-Tayaran A, Al-Jubair AH, Gomaa A. Trends in water quality of some wells in Saudi Arabia, 1984-1989. *Sci Total Environ.* 1994;143(2-3):173-181.

Al-Ibrahim AA. Excessive use of groundwater resources in Saudi Arabia: impacts and policy options. *Ambio.* 1991;20:34-37.

APHA, AWWA, WPCF. *Standard Methods for the Examination of Water and Wastewater. 15th ed.* Washington, DC: American Public Health Association; 1980.

Ashkenazi S, Bellah G, Cleary TG. Hallucinations as an initial manifestation of childhood shigellosis. *J Pediatr.* 1989;114:95-96.

Gastrointestinal Disease

Echeverria P, Ramirez G, Blacklow NR, Ksiazek T, Cukor G, Cross JH. Travelers' diarrhea among U.S. Army troops in South Korea. *J Infect Dis.* 1979;139:215-219.

Echeverria P, Seriwatana I, Sethabutr O, Chatkaeomorakot A. Detection of diarrheogenic *Escherichia coli* using nucleotide probes. In: Macario A, Conway de Macario E, eds. *Gene Probes for bacteria.* New York, Ny: Academic Press; 1990:95-141.

Ericsson CD, DuPont HL, Mathewson JJ, West MS, Johnson PC, Bitsura JM. Treatment of traveler's diarrhea with sulfamethoxazole and trimethoprim and loperamide. *JAMA.* 1990;263:257-261.

Ericsson CD, Johnson PC, DuPont HL, Morgan DR, Bitsura JAM, de la Cabada FJ. Ciprofloxacin or trimethoprim-sulfamethoxazole as initial therapy for travelers' diarrhea: a placebo-controlled, randomized trial. *Ann Intern Med.* 1987;106:216-220.

Farid Z, Bassily S, Kent DC, et al. Chronic urinary *salmonella* carriers with intermittent bacteraemia. *J Trop Med Hyg.* 1970;73:153-156.

Forslund J. Groundwater quality today and tomorrow. *W H Stat Quat.* 1986;39:81-90.

Fried JJ. *Groundwater Pollution.* The Netherlands: Elsevier Scientific Publishing Co.; 1975.

Gary GW, Anderson LJ, Keswick BH, et al. Norwalk virus antigen and antibody response in an adult volunteer study. *J Clin Microbiol.* 1987;25:2001 -2003.

Gilman RH, Terminel M, Levine MM, Hernandez-Mendoza P, Hornick RB. Relative efficacy of blood, urine, rectal swab, bone-marrow, and rose spot cultures for recovery of *Salmonella typhi* in typhoid fever. *Lancet* 1975;1:1211-1213.

Gotuzzo E, Oberhelman RA, Maguina C, et al. Comparison of single-dose treatment with norfloxacin and standard 5-day treatment with trimethoprim-sulfamethoxazole for acute shigellosis in adults. *Antimicrob Agents Chemother.* 1989;33:1101-1104.

Guerra-Caceres JG, Gotuzzo-Herencia E, Crosby-Dagnino E. Miro-Quesada M, Carrillo-Parodi C. Diagnostic value of bone marrow culture in typhoid fever. *Trans R Soc Trop Med Hyg.*1979;73:680-683.

Gunn RA, Kimball AM, Mathew PP, Dutta SR, Rifaat AH. Cholera in Bahrain: epidemiological characteristics of an outbreak. *Bull World Health Organ.* 1981;59:61-66.

Haberberger RL, Thornton SA, Scott DA, Hyams KC. Diarrheal disease aboard a U.S. Navy ship after a brief port visit to a high risk area. *Mil Med.* 1994;159:445-448.

Haberberger RL, Diniega BM, Mikhail IA, et al. Travelers' diarrhea among United States military personnel during joint American-Egyptian Armed Forces Exercises in Cairo, Egypt. *Mil Med.* 1991;156:27-30.

Halpern Z, Dan M, Giladi M, et al. Shigellosis in adults: epidemiologic, clinical, and laboratory features. *Medicine.* 1989;68:210-217.

Hathout SE, El-Ghaffar YA, Awny AY. Salmonellosis complicating schistosomiasis in Egypt: a new clinical appreciation. *Am J Trop Med Hyg.* 1967;16:162-172.

Helin I, Araj GF. Antibiogram of urinary tract isolates in Kuwait. *Scand J Infect Dis.* 1986;18:447-450.

Hoffman SL, Punjabi NH, Kumala S, et al. Reduction of mortality in chloramphenicol-treated severe typhoid fever by high-dose dexamethasone. *N Engl J Med.* 1984;310:82-88.

Hyams KC, Bourgeois AL, Merrell BR, et al. Diarrheal disease during Operation Desert Shield. *N Engl J Med.* 1991;325:1423-1428.

Islam A, Butler T, Nath SK, et al. Randomized treatment of patients with typhoid fever by using ceftriaxone or chloramphenicol. *J Infect Dis.* 1988;158:742-747.

Ismail AMA. The quality of irrigation groundwater in Qatar and its effect on the development of agriculture. *J Arid Environ.* 1984;7:101-106.

Kasim AA, Elhelu MA. Giardiasis in Saudi Arabia. *Acta Trop (Basel).* 1983;40:155-158.

Kassimi MA, Gosling PI, Khogheer YA. Trends in bacterial gastroententis in the western region of Saudi Arabia. *J Hosp Infect.* 1985;6:234-235.

Keusch GT, Bennish ML. Shigellosis: recent progress, persisting problems, and research issues. *Pediatr Infect Dis J.* 1989;8:713-719.

Khuffash FA, Majeed HA, Sethi SK, Al-Nakib W. Gastroententis in a regional hospital in Kuwait: some aspects of the disease. *Ann Trop Paediatr.* 1982;2:123-128.

Gastrointestinal Disease

Kligler RM, Hoeprich PD. Shigellemia. *West J Med.* 1984;141:375-378.

Lakshmanan AR, Krishna TR, Viswanathan S. Nitrate and fluoride levels in drinking waters in the Twin cities of Hyderabad and Secunderabad. *Indian J Environ Health.* 1986;28:39-47.

Langenegger O. High nitrate concentration in shallow aquifers in a rural area of central Nigeria caused by random deposits of domestic refuse and excrement. In: Duijvenbooden WV, Alasbergen P, Lelyveld HV, eds. *Quality of Groundwater.* The Netherlands: Elsevier Scientific Publishing Co.; 1981: 135-140.

Lew JF, Kapikian AZ, Jiang X, Estes MK, Green KY. Molecular characterization and expression of the capsid protein of a Norwalk-like virus recovered from a Desert Shield troop with gastroenteritis. *Virology.* 1994;200:319-325.

Martin DL, Gustafson TL, Pelosi JW, Suarez L, Pierce GV. Contaminated produce—a common source for two outbreaks of *Shigella* gastroenteritis. *Am J Epidemiol.* 1986;124:299-305.

Mikhail IA, Fox E, Haberberger RL Jr, Ahmed MH, Abbattee EA. Epidemiology of bacterial pathogens associated with infectious diarrhea in Djibouti. *J Clin Microbiol* 1990;28:956-961.

Mikhail IA, Higashi GI, Edman DC, Elwan SH. Interaction of *Salmonella paratyphi* A and *Schistosoma mansoni* in hamsters. *Am J Trop Med Hyg.* 1982;31:328-334.

Mimouni O, Chibane B. Predicting nitrate pollution of Mitidja Plain groundwater. *Environ Software.* 1989;4:136-141.

Murray BE. Resistance of *Shigella, Salmonella,* and other selected enteric pathogens to antimicrobial agents. *Rev J Infect Dis.* 1986;8(suppl 2):S172-S181.

Neves J, Marinho RP, Lobo Martins NR da Luz, De Araujo PK, Lucciola J. Prolonged septicaemic salmonellosis: treatment of intercurrent schistosomiasis with niridazole. *Trans R Soc Trop Med Hyg 1969.* 1969;63:79-84.

Neves J, Martins NR. Long duration of septicaemic salmonellosis: 35 cases with 12 implicated species of *Salmonella. Trans R Soc Trop Med Hyg.* 1967;61:541-552.

Oldfield EC III, Bourgeois AL, Omar AM, Pazzaglia GL. Empirical treatment of *Shigella* dysentery with trimethoprim: five-day course vs. single dose. *Am J Trop Med Hyg.* 1987;37:616-623.

Oprandy JJ, Thornton SA, Gardiner CH, Burr D, Batchelor R, Bourgeois AL . Alkaline phosphatase-conjugated oligonucleotide probes for enterotoxigenic *Escherichia coli* in travelers to South America and West Africa. *J Clin Microbiol.* 1988;26:92-95.

Paparello SF, Bourgeois AL, Garst P, Hyams KC. Diarrheal and respiratory disease aboard the hospital ship, USNS Mercy T-AH 19, during Operation Desert Shield. *Mil Med.* 1993;158:392-395.

Pazzaglia G, Walker RI. A retrospective survey of enteric infections in active duty Navy and Marine Corps personnel. *Mil Med.* 1982;147:27-33.

Philbrook FR, Gordon JE. Diarrhea and Dysentery. In: Hoff EC, ed. *Preventive Medicine in World War II. Vol. 4. Communicable Diseases.* Washington, DC: Office of the Surgeon General, U.S. Department of the Army; 1958:319-376.

Qadri MH, Al-Ghamdi MA, Imadulhaq M. Acute diarrheal disease in children under five years of age in the Eastern Province of Saudi Arabia. *Annals of Saudi Medicine.* 1990;10:280-284.

Raveendran E, Madany IM. The quality of goundwater in Bahrain. *Sci Total Environ.* 1991;103:177-184.

Rocha H, Kirk JW, Hearey CD. Prolonged *Salmonella* bacteremia in patients with *Schistosoma mansoni* infection. *Arch Intern Med.* 1971;128:254-257.

Sack DA, Kaminsky DC, Sack RB, et al. Enterotoxigenic *Escherichia coli* diarrhea of travelers: a prospective study of American Peace Corps volunteers. *Johns Hopkins Med J.* 1977;141:63-70.

Salih SY, Subaa HA, Asha HA, Satir AA. Salmonellosis complicating schistosomiasis in the Sudan. *J Trop Med Hyg.* 1977;80:14-18.

Scott DA, Haberberger RL, Thornton SA, et al. Norfloxacin for the prophylaxis of travelers' diarrhea in U.S. military personnel. *Am J Trop Med Hyg.* 1990;42:160-164.

Sethi SK, Khuffash FA. Bacterial and viral causes of acute diarrhoea in children in Kuwait. *J Diarrhoeal Dis Res.* 1989;7:85-88.

Sethi SK, Khuffash FA, al-Nakib W. Microbial etiology of acute gastroenteritis in hospitalized children in Kuwait. *Pediatr Infect Dis J*. 1989;8:593.

Sharp TW, Thornton SA, Wallace MR, et al. Diarrheal disease among military personnel during Operation Restore Hope, Somalia, 1992-1993. *Am J Trop Med Hyg*. 1995;52:188-193.

Taylor DN, Sanchez JL, Candler W, Thornton S, McQueen C, Echeverria P. Treatment of travelers' diarrhea: ciprofloxacin plus loperamide compared with ciprofloxacin alone: a placebo-controlled, randomized trial. *Ann Intern Med*. 1991;114:7314.

Taylor WR, Schell WL, Wells JG, et al. A foodborne outbreak of enterotoxigenic *Escherichia coli* diarrhea. *N Engl J Med*. 1982;306:1093-1095.

Tell S, Sara Y. *State of the Environment in Jordan*. Jordan: Ministry of Municipal and Rural Affairs and the Environment; 1989.

Wallace M, Yousif AA. Spread of multiresistant *Salmonella typhi*. *Lancet*. 1990;336:1065-1066.

Williams HMS, Richards J. Single-dose ciprofloxacin for shigellosis [letter]. *Lancet*. 1990;335:1343-1344.

Wolf MK, Taylor DN, Boedeker EC, et al. Characterization of enterotoxigenic *Escherichia coli* isolated from U.S. troops deployed to the Middle East. *J Clin Microbiol*. 1993;31:851-856.

Yousif AA, Alla AQ, Daniels M, Fernandes EL. Shigellosis: species prevalence, incidence, and drug resistance in Bahrain. *Journal of the Bahrain Medical Society*. 1989;1:52-55 .

Yusop MK, Cleemput OV, Baert L. Nitrogen fertilization and nitrate pollution of groundwater in Sandy Soils. *Environ Pollut (Series B)*. 1984;7:43-48.

Zajdowicz T. Epidemiologic and clinical aspects of shigellosis in American forces deployed to Saudi Arabia. *South Med J*. 1993;86:647-50.

INSECTICIDES

Abou-Donia MB. Biochemical toxicology of organophosphorous compounds. In: Blum K, Manzo L, eds. *Neurotoxicology*. New York, Ny: Marcel Dekker;1985:423-444.

Abou-Donia MB. The cytoskeleton as a target for organophosphorus ester-induced delayed neurotoxicity (OPIDN). *Chem Biol Interact*. 1993;87(1-3):383-393.

Aldridge N. Postscript to the symposium on organophosphorus compound induced delayed neuropathy. *Chem Biol Interact*. 1993;87(1-3):463-466.

Aldridge WN. An assessment of the toxicological properties of pyrethroids and their neurotoxicity. *Crit Rev Toxicol*. 1990;21:89-104.

Ames RG, Brown SK, Mengle DC, Kahn E, Stratton JW, Jackson RJ. Protecting agricultural applicators from over-exposure to cholinesterase-inhibiting pesticides: perspectives from the California programme. *J Soc Occup Med*. 1989;39(3):85-92.

Anadon A, Martinez-Larranaga MR, Diaz MJ, Bringas P. Toxicokinetics of permethrin in the rat. *Toxicol Appl Pharmacol*. 1991;110:1-8.

Anderson D, Richardson CP. *Permethrin (PP557): Cytogenetic study in the rat*. CTL/P/294. Imperial Chemical Industries Ltd; 1976.

Andrews EB, Joseph MC, Magenheim MJ, Tilson HH, Doi PA, Schultz MW. Postmarketing surveillance study of permethrin creme rinse. *Am J Public Health*. 1992;82:857-861.

Barrett DS, Oehme FW, Kruckenberg SM. A review of organophosphorus ester-induced delayed neurotoxicity. *Vet Hum Toxicol*. 1985;27(1):22-37.

Barrueco C, Herrera A, Caballo C, de la Pena E. Cytogenic effects of permethrin in cultured human lymphocytes. *Mutagenesis*. 1992;7;433-437.

Barrueco C, Herrera A, Caballo C, de la Pena E. Introduction of structural chromosome aberrations in human lymphocyte cultures and CHO cells by permethrin. *Teratogenesis, Carcinog, Mutagen*. 1994;14:31-38.

Bartelt N, Hubbel J. Percutaneous absorbtion of topically applied ^{14}C-permethrin in volunteers: final medical report. *Borroughs Wellcome Co*. 1987.

Ben-Dyke R, Toscano MJ, Ventura P, Shapiro R. Permanone tick repellent: an acute 4-hour inhalation toxicity study in rats. *Product Safety Labs, Inc*. 1987.

Insecticides

Bloomquist L, Thorsell W. Distribution and fate of the insect repellent 14C-N, N-diethyl-meta-toluamide in the aminal body. II. Distribution and excretion after cutaneous application. *Acta Pharmacol Toxicol.* 1977;41:235-243.

Bokonjic D, Rosie N. Anticonvulsive and protective effects of diazepam and midazolam in rats poisoned by highly toxic organophosphorus compounds. *Arhic Az Higijenu Rada I Toksikologiju.* 1991;42:359-365.

Bowerman JG, Gomez MP, Austin RD, Wold DE. Comparative study of permethrin 1% creme rinse and lindane shampoo for the treatment of head lice. *Pediatr Infect Dis J.* 1987;6:252-255.

Brammer A. *Permethrin: 4-Hour Acute Inhalation Toxicity Study in the Rat.* CTL/P/2492. Imperial Chemical Industries Ltd; 1989.

Brandenburg K, Deinard AS, DiNapoli J, Englender SJ, Orthoefer J, Wagner D. 1% permethrin cream rinse vs. 1% lindane shampoo in treating Pediculosis capins. *Am J Dis Child.* 1986;140:894-896.

Breeden GC, Schreck CE, Sorensen AL. Permethrin as a clothing treatment for personal protection against chigger mites (Acarina: Trombiculidae). *Am J Trop Med Hyg.* 1982;3:589-592.

Bright JE, Inns RH, Tuckwell NJ, Griffiths GD, Marrs TC. A histochemical study of changes observed in the mouse diaphragm after organophosphate poisoning. *Hum Exp Toxicol.* 1991;10:9-14.

Brini CM, Tremblay GC. Reversible inhibition of the urea cycle and gluconeogenesis by N, N-diethyl-m-toluamide. *Biochem Biophys Res Commun.* 1991;179(3):1264-1268.

Burchfiel JL, Duffy FH. Organophosphate neurotoxicity: chronic effects of sarin on the electroencephalogram of monkey and man. *Neurobehavioral Toxicol Teratol.* 1982;4:767-778.

Butterworth STG, Hend RW. *Toxicity Studies on the Insecticide WL43470: A Five Week Feeding Study in Rats.* TLGR.0056.76. Shell Research Ltd; 1976.

Callander RD. *Permethrin: An Evaluation in the Salmonella Mutation Assay.* CTL/P/2423. Imperial Chemical Industries Ltd; 1989.

Carjuzaa A, Carpentier JP. Myoresolution: organophosphorus nerve agents and chemical warfare agents: curare problems. *Med Armees.* 1991;19(2):97-98.

Casida JE, Gammon DW, Glickman AH. Mechanisms of selective action of pyrethroid insecticides. *Annu Rev Pharmacol Toxicol.* 1983;23:413-438.

Casida JE, Ueda K, Gaughan LC, Jao LT, Soderlund DM. Structure-biogradability relationships in pyrethroid insecticides. *Arch Environ Contam Toxicol.* 1976;3:491-500.

CEPA (California Environmental Protection Agency). *Permethrin (Permanone Tick Repellent) Risk Characterization Document.* Medical Toxicology and Worker Health and Safety Branches, Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, Calif; 1992.

Cherniack MG. Toxicological screening for organophosphorus-induced delayed neurotoxicity: complications in toxicity testing. *Neurotoxicology.* 1988;9(2):249-271.

Chesher BC, Malone IC. *Ocular irritancy of 21Z73 in rabbits.* HEFG 74-6. Wellcome Research Laboratories; 1974.

Chesher BC, Malone IC. *Guinea pig sensitisation study with 21Z73 using the maximization test method.* HEFG 74-3. Wellcome Research Laboratories; 1974.

Chesher BC, Clampitt RC, Malone IC. *21Z73—Preliminary into the cumulative oral toxicity on dogs.* HEFG 75-9. Wellcome Research Laboratories; 1975.

Chesher BC, Malone IC, Parker MI. *21Z73. Dominant lethal study in male mice.* HEFG 75-10. Wellcome Research Laboratories; 1975.

Clapp MIL, Banham PB, Glaister IR, Moyes A. *PP557: 28 day feeding study in mice.* CTLJP/354. Imperial Chemical Industries Ltd; 1977.

Clapp MIL, Banham PB, Chart IS, Glaister J, Gore C, Moyes A. *PP557: 28 day feeding study in rats.* CTL/P/385. Imperial Chemical Industries Ltd; 1977.

Coats JR. Mechanisms of toxic action and structure-activity relationships for organochlorine and synthetic pyrethroid insecticides. *Environ Health Perspect.* 1990;87:255-262.

Insecticides

Dayan AD. *21-day neuropathological study in the Sprague Dawley rat of permethrin (21Z73ZJ) administered in the diet.* BPAT 80/48. Wellcome Research Laboratories; 1980.

De Bleecker JL, De Reuck JL, Willems JL. Neurological aspects of organophosphate poisoning. *Clin Neurol Neurosurg.* 1992;94(2):93-103.

Dees WH, Overington KR, Karuru ER, Helmy I, Schreck CE. *Preliminary investigations of protective clothing and repellents used against medically important arthropods in the Sinai Desert [abstract].* Proceedings of the American Mosquito Control Association and New Jersey Mosquito Control Association Meetings; 1986.

deGarbino JP, Laborde A. Toxicity of an insect repellent N-N-diethyltoluamide. *Vet Hum Toxicol.* 1983;25:422-423.

Dellinger JA. Monitoring the chronic effects of anticholinesterase pesticides in aerial applicators. *Vet Hum Toxicol.* 1985;27(5):427-430.

DiNapoli IB, Austin RD, Englender SJ, Gomez MP, Barrett IF. Eradication of head lice with a single treatment. *Am J Public Health.* 1988;78:978-980.

Dorman DC. Diethyltoluamide (DEET) insect repellent toxicosis. *Vet Clin North Am Small Anim Pract.* 1990;20(2):387-391.

Duffy FH, Burchfiel JL, Bartels PH, Gaon M, Sim VM. Long-term effects of an organophosphate upon the human electroencephalogram. *Toxicol Appl Pharmacol.* 1979;47:161-176.

Dyck PJ, Shimono M, Schoening GP, Lais AC, Oviatt KF, Sparks MF. The evaluation of a new synthetic pyrethroid pesticide (permethrin) for neurotoxicity. *J Environ Pathol Toxicol Oncol.* 1984;5:109-117.

Edwards DL, Johnson E. Insect repellent induced toxic encephalopathy in a child. *Clin Pharm.* 1987;6:496-498.

Edwards MJ, Iswaran TJ. *Permethrin: Residue Transfer and Toxicology Study With Cows Fed Treated Grass Nuts.* TMJ1519/B. Imperial Chemical Industries Ltd; 1977.

Eells JT, Bandettini PA, Holman PA, Propp JM. Pyrethroid insecticide-induced alterations in mammalian synaptic membrane potential. *J Pharmacol Exp Ther.* 1992;262:1173-1181.

Eil C, Nisula BC. The binding properties of pyrethroids to human skin fibroblast androgen receptors and to sex hormone binding globulin. *J Steroid Biochem.* 1990;35:409-414.

EPA. *Permethrin— Quantitative risk assessment, two-year chronic/oncogenicity mouse (females) study.* Memo from Bernice Fisher to John Doherty, Health Effects Division, Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.; Dec. 23, 1988.

EPA. *Peer review of permethrin.* Memo from Esther Rinde, Health Effects Division, to George LaRocca, Registration Division, Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, DC; April 7, 1989.

EPA. *Second peer review of permethrin—Consideration of recommendations by the scientific advisory panel regarding the oncogenic classification of permethrin.* Memo from Edwin R. Budd, Health Effects Division, to George LaRocca, Registration Division, Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.; Sept. 18, 1989.

EPA. *Evaluation of exposure for military uses of permethrin tick repellant.* HED Project No. 0-1507. Memo from Curt Lunchick, Health Effects Division, to George LaRocca, Registration Division, Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.; July 30, 1990.

EPA. *Insect/arthropod repellent fabric treatment formulations containing permethrin for military use.* Memo from Roger Gardner, Health Effects Division, to George LaRocca, Registration Division, Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.; December, 1990.

Erhard MH, Kuchlmann R, Szimicz L, Loesch U. Detection of the organophosphorus nerve agent soman by an ELISA using monoclonal antibodies. *Arch Toxicol.* 1990;64(7):580-585.

Evans SR, Korch GW Jr, Lawson MA. Comparative field evaluation of permethrin and DEET-treated military uniforms for personal protection against ticks (*Acari*). *J Med Entomol.* 1990;27(5):829-834.

Farquhar JA, Hutchinson DBA, Periam AW, Sparks RG. *An Investigation Into the Absorption of Permethrin From Impregnated Clothing.* BKPL/81/1. Wellcome Research Laboratories; 1981.

Flannigan SA, Tucker SB. Variation in cutaneous sensation between synthetic pyrethroid insecticides. *Contact Dermatitis* 1985;13:140-147.

Flannigan SA, Tucker SB, Key MM, et al. Primary irritant contact dermatitis from synthetic pyrethroid insecticide exposure. *Arch Toxicol.* 1985;56:288-294.

Flannigan SA, Tucker SB, Marcus MK, et al. Synthetic pyrethroid insecticides: a dermatological evaluation. *Br J Ind Med.* 1985;42:363-372.

Garrett NE, Stack HF, Waters MD. Evaluation of the genetic activity profiles of 65 pesticides. *Mutat Res.* 1986;168(3):301-326.

Gaughan LC, Unai T, Casida JE. Permethrin metabolism in rats. *J Agric Food Chem.* 1977;25:9-17.

Glaister JR, Pratt I, Richards D. *Effects of high dietary levels of PP557 on clinical behavior and structure of sciatic nerves in rats.* CTL/P/317. Imperial Chemical Industries Ltd; 1977.

Glickman AH, Hamid AAR, Rickert DE, Lech JJ. Elimination and metabolism of permethrin isomers in rainbow trout (*Salmo gairdneri*). *Toxicol Appl Pharmacol.* 1981;57:88-98.

Glickman AH, Lech JJ. Hydrolysis of permethrin, a pyrethroid insecticide, by rainbow trout (*Salmo gairdneri*) and mouse tissues in vitro: A comparative study. *Toxicol Appl Pharmacol.* 1981;60:186-192.

Gupta RK, Rutledge LC. Compatibility of Army face paint and insect repellent formulation. *Mil Med.* 1993;158(1):12-13.

Gutmann L, Besser R. Organophosphate intoxication: pharmacologic, neurophysiologic, clinical, and therapeutic considerations. *Semin Neurol.* 1990;10(1):46-51.

Hart D, Banham PB, Glaister JR, Pratt I, Weight TM. *PP557: Whole Life Feeding Study in Mice.* CTL/P/359. Imperial Chemical Industries Ltd; 1977.

He F, Wang S, Liu L, Chen S, Zhang Z, Sun J. Clinical manifestations and diagnosis of acute pyrethroid poisoning. *Arch Toxicol.* 1989;63:54-58.

Heick HMC, Peterson RG, Dalpe-Scott M, Qureshi IA. Insect repellent, *N,N*-Diethyl-*m*-toluamide, effect on ammonia metabolism. *Pediatrics.* 1988;82:373-376.

Hend RW, Butterworth STG. *Toxicity of Insecticides: A Short-Term Feeding Study of WL 43379 in Rats.* TLGR.0108.77. Shell Research Ltd; 1977.

Herrera A, Barrueco C, Caballo C, de la Pena E. Effect of permethrin on the induction of sister chromatid exchanges and micronuclei in cultured human lymphocytes. *Environ Mol Mutagen.* 1992;20:218-222.

Hierons R, Johnson MK. Clinical and toxicological investigations of a case of delayed neuropathy in man after acute poisoning by an organophosphorus pesticide. *Arch Toxicol.* 1978;40(4):279-284.

Hodge MCE, Banham PB, Glaister JR, Richards D, Taylor K, Weight TM. *PP557: Three-Generation Reproduction Study in Rats.* CTL/P/361. Imperial Chemical Industries Ltd; 1977.

Hodge MCE. *Permethrin: Teratogenicity Study in the Rat.* CTL/P/2269. Imperial Chemical Industries Ltd; 1988.

Hoellinger H, Lecorsier A, Do-Cao-Thang. The micronucleus test for possible cytogenotoxicity of some pyrethroids. *Mutat Res.* 1984;130:244.

Hoellinger H, Lecorsier A, SoMier M, Leger C, Do-Cao-Thang, and Nguyen-Hoang-Nam. Cytotoxicity, cytogenotoxicity, and allergenicity tests on certain pyrethroids. *Drug Chem Toxicol.* 1987;10:291-310.

Hutson DH. The metabolic fate of synthetic pyrethroid insecticides in mammals. *Prog Drug Metab.* 1979;3:215-252.

Inns RH, Tuckwell NJ, Bright JE, Marrs TC. Histochemical demonstration of calcium accumulation in muscle fibres after experimental organophosphate poisoning. *Hum Exp Toxicol.* 1990;9:245-250.

IPCS (International Programme on Chemical Safety). *Permethrin.* Environmental Health Criteria 94. Series 94. Geneva: World Health Organization; 1990.

Ishmael JE. *Permethrin: Acute Oral Toxicity to the Rat.* CTL/P/2453. Imperial Chemical Industries Ltd; 1989.

Ishmael J, Litchfield MH. Chronic toxicity and carcinogenic evaluation of permethrin in rats and mice. *Fundam Appl Toxicol.* 1988;11:308-322.

Insecticides

McCreesh AH. *Toxicological Evaluation of 3-(phenoxyphenyl) methyl (+)-cis,trans-3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylate (Permethrin).* Study No. 51-0831-78. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1977.

Mehr ZA, Korte DW, Justus ID, Gupta RK. *Mutagenic Potential of Permethrin in the Drosophila Melanogaster Six Linked Recessive Lethal Test.* Letterman Army Institute Research Rep. 302. Letterman Army Institute, San Francisco, Ca. NTIS No. AD-A201802/6/XAB; 1988.

Metker LW. *Subchronic Inhalation Toxicity of 3 (phenoxyphenyl)methyl(+)-cis,trans-3-(2,2-dichloroethenyl)-2,2 dimethylcyclopropanecarboxylate (Permethrin).* 75-51-0026-80. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1978.

Metker LW, Angerhofer RA, Pope CR, Swentzel KC. *Toxicology Evaluation of 3-(phenoxyphenyl)methyl(+)-cis,trans-3 (2,2-dichloroethenyl)-2,2-demethylcyclopropanecarboxylate (Permethrin).* 75-51-0837-78. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1977.

Millner CK, Butterworth STG. *Toxicity of Pyrethroid Insecticides: Investigation of the Neurotoxic Potential of WL 43479 to Adult Hens.* TLGR.0069.77. Shell Research Ltd; 1977.

Minton NA, Murray VS. A review of organophosphate poisoning. *Med Toxicol Adverse Drug Exp.* 1988;3(5):350-375.

Miyamoto J. Degradation, metabolism and toxicity of synthetic pyrethroids. *Environ Health Perspect.* 1976;14:15-28.

Moody RP, Riedel D, Ritter L, Franklin CA. The effect of DEET (*N,N*-diethyl-*m*-toluamide) on dermal persistence and absorption of the insecticide fenitrothion in rats and monkeys. *J Toxicol Environ Health* 1987;22:471-479.

Mount GA, Snoddy EL. Pressurized sprays of permethrin and DEET on clothing for personal protection against the Lone Star tick and the American dog tick (Acari: Ixodidae). *J Econ Entomol.* 1983;76:529-531.

National Research Council. Health Effects of Permethrin-Impregnated Army Battle-Dress Uniforms. National Research Council, Commission on Life Sciences, Board on Environmental Studies and Toxicology, Committee on Toxicology, Subcommittee to Review Permethrin Toxicity From Military Uniforms. Washington, D.C.: National Academy Press; 1994.

Nelson DC. *Studies on the combined toxicity of DEET and permethrin.* Report to the Repellents Committee, Armed Forces Pest Management Board; 1989.

NRCC (National Research Council of Canada). Pyrethroids: *Their effects on aquatic and terrestrial ecosystems.* NRCC No. 24376. Ottawa, Ontario: National Research Council of Canada; 1986.

Okuno Y, Miyagawa H, Hosoi R, Kadota T. *Skin and Eye Irritation Study of Permethrin (S-3151) in Rabbits.* Sumitomo Chemical Co; 1976.

Okuno Y, Kadota T, Miyamoto J. *Neurotoxic Effects of Some Synthetic Pyrethroids By Short-Term Feeding in Rats.* AT-60 0046. Sumitomo Chemical Co; 1976.

Osimitz TG, Grothaus RH. The present safety assessment of DEET. *J Am Mosq Control Assoc.* 1995;11(2):274-278.

Paldy A. Examination of the mutagenic effect of synthetic pyrethroids on mouse bone marrow cells. *Proc Hung Annu Meet Biochem.* 1981;21:227-228.

Parkinson GR. *Permethrin: Acute Toxicity to Male Rats.* CTL/P/388. Imperial Chemical Industries Ltd; 1978.

Parkinson GR, Berry PN, Glaister J, Gore CW, Lefevre VK, Murphy IA. *PP557 (Permethrin): Acute and Sub-Acute Toxicity.* CTL/P/225. Imperial Chemical Industries Ltd; 1976.

Pegum IS, Doughty BJ. *Human Skin Patch Tests With Pyrethrins, Kadethrin, Permethrin, and Decamethrin.* NEFI 78-2. Wellcome Research Laboratories; 1978.

Pluijmen M, Drevon C, Montesano R, Malaveille C, Hautefeuille A, Bartsch H. Lack of mutagenicity of synthetic pyrethroids in *Salmonella typhimurium* strains and in V79 Chinese hamster cells. *Mutat Res.* 1984;137:7-15.

Prinsen GH, van Sittert NJ. *Exposure and Medical Monitoring Study of the Pyrethroid WL 43479 After Single Application for Stable Fly Control in West Germany.* Rep. Ser. Tox 78-005. Shell Chemical Co; 1978.

Rengstorff RH. Vision and ocular changes following accidental exposure to organophosphates. *J Appl Toxicol.* 1994;14:115-118.

Insecticides

IUPAC (International Union of Pure and Applied Chemistry). The chemistry, metabolism and residue analysis of synthetic pyrethroids. *Pure Appl Chem.* 1981;53:1967-2022.

Jaggers SE, Parkinson GR. *Permethrin: Summary and Review of Acute Toxicities in Laboratory Species.* CTL/P/461. Imperial Chemical Industries Ltd; 1979.

James DA. *Foetal Toxicity Study of 21Z73 (NRDC 143) in the Rat.* BPAT 74/10. Wellcome Research Laboratories; 1974.

James DA. *Foetal Toxicity Study of 21Z73 (NRDC 143) in the Mouse.* BPAT 74/12. Wellcome Research Laboratories; 1974.

James DA. *Foetal Toxicity Study of 21Z73 (NRDC 143) in the Rabbit.* BPAT 74/19. Wellcome Research Laboratories; 1974.

James DA. *A Multigeneration Reproduction Study of 21Z73 (Permethrin) in the Rat.* BPAT 79-3. Wellcome Research Laboratories; 1979.

James JA, Taylor PE, Roe FJC. *Carcinogenicity Study in Mice With Permethrin (21Z73).* Wellcome Research Laboratories; 1980.

Karalliedde L, Henry JA. Effects of organophosphates on skeletal muscle. *Hum Exp Toxicol.* 1993;12(4):289-296.

Klette KL, Levine B, Dreka C, Smith ML, Goldberger BA. Cholinesterase activity in postmortem blood as a screening test for organophosphate chemical weapon exposure. *J Forensic Sci.* 1993;38(4):950-955.

Kohda H, Kadota T, Miyamoto J. *Teratogenic Evaluation with Permethrin in Rats.* IT-60-0102. Sumitomo Chemical Co; 1976.

Kohda H, Kadota T, Miyamoto J. *Teratogenic Evaluation with Permethrin in Mice.* IT-60-0101. Sumitomo Chemical Co; 1976.

Kohda H, Kadota T, Miyamoto J. *Acute Oral, Dermal and Subcutaneous Toxicities of Permethrin in Rats and Mice.* Sumitomo Chemical Co; 1979.

Kolmodin-Hedman B, Swensson A, Akerblom M. Occupational exposure to some synthetic pyrethroids (permethrin and fenvalerate). *Arch Toxicol.* 1982;50:27-33.

Lau WM, Szilagyi M, Freeman SE. Effects of some organophosphorus compounds on the binding of a radioligand (8-cyclopentyl 1, 3-[3H]dipropylxanthine) to adenosine receptors in ovine cardiac membranes. *J Appl Toxicol.* 1991;11:411-414.

Leah AM. *Permethrin: Eye Irritation to the Rabbit.* CTLIP/2455. Imperial Chemical Industries Ltd; 1989.

Leah AM. *Permethrin: Skin Sensitization Study.* CTL/P/22456. Imperial Chemical Industries Ltd; 1989.

Le Quesne PN, Maxwell IC, Butterworth TG. Transient facial sensory symptoms following exposure to synthetic pyrethroids: a clinical and electrophysiological assessment. *Neurotoxicology.* 1980;2:1-11.

Life Science Research. *Chronic Feeding Study of Permethrin in the Rat.* Wellcome Research Laboratories; 1980.

Lillie TH, Schreck CE, Rahe AJ. Effectiveness of personal protection against mosquitoes in Alaska. *J Med Entomol.* 1988;25:475-478.

Litton Bionetics, Inc. *Mutagenicity Evaluation of Permethrin.* Litton Bionetics, Inc; 1975.

Longstaff E . *Permethrin: Short-Term Predicitive Tests for Carcinogenicity. Results From the Ames Test.* CTL/P/301. Imperial Chemical Industries Ltd; 1976.

Lotti M. Biological monitoring for organophosphate-induced delayed polyneuropathy. *Toxicol Lett.* 1986;33(1-3):167-172.

Lotti M. The pathogenesis of organophosphate polyneuropathy. *Crit Rev Toxicol.* 1991;21(6):465-487.

Maddy KT, Edmiston S, Richmond D. Illness, injuries, and deaths from pesticide exposures in California, 1949-1988. *Rev Environ Contam Toxicol.* 1990;114:57-123.

Mahieu P, Lauwerys R, Dive A, Hantson P. Poisoning by some insecticides, herbicides and fungicides. *Acta Clin Belg Suppl.* 1990;13:75-85.

Marei AEM, Ruzo LO, Casida JE. Analysis and persistence of permethrin, cypermethrin, deltamethrin, and fenvalerate in the fat and brain of treated rats. *J Agric Food Chem.* 1982;30:558-562.

McCorkle F, Taylor R, Martin D, Glick B. The effect of permethrin on the immune response of chickens. *Poult Sci.* 1980;59:1568. Abstract

Insecticides

Shapiro R. *Permanone Multi-Use Insecticide Spray: EPA Acute Oral Toxicity Limit Test.* Product Safety Labs, Inc; 1989.

Sherman RA. *Preliminary Behavioural Assessment of Habituation to the Insecticide Permethrin.* 75-51-002679. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1979.

Sholdt LL, Schreck CE, Mwangelwa MI, Nondo J, Siachinji VJ. Evaluations of permethrin-impregnated clothing and three topical repellent formulations of DEET against tsetse flies in Zambia. *Med Vet Entomol.* 1989;3:153-158.

Sholdt LL, Schreck CE, Qureshi A, Mammino S, Aziz A, Iqbal M. Field bioassays of permethrin-treated uniforms and a new extended duration repellent against mosquitoes in Pakistan. *J Am Mosq Control Assoc.* 1988;3:233-236.

Sidon EW, Moody RP, Franklin CA. Percutaneous absorption of cis- and trans-permethrin in rhesus monkeys and rats: anatomic site and interspecies variation. *J Toxicol Environ Health* 1988;23:207-216.

Snodgrass HL. Permethrin transfer from treated cloth to the skin surface: potential for exposure in humans. *J Toxicol Environ Health.* 1992;35(2):91-105.

Snodgrass HL Jr. Skin sensitization of the insecticide permethrin in man and the potential for nonimmunological contact urticaria. Study No. 15-51-0351-86. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1986.

Spencer F, Berhance Z. Uterine and fetal characteristics in rats following a post-implantational exposure to permethrin. *Bull Environ Contam Toxicol.* 1982;29:84-88.

Stelzer KJ, Gordon MA. Effects of pyrethroids on lymphocyte mitogenic responsiveness. *Res Commun Chem Pathol Pharmacol.* 1984;46(1):137-150.

Taplin D, Meinking TL. *Scabies and Lice: An Update for the 90's.* Paper presented at the 53rd Annual Meeting of the American Academy of Dermatology. Washington, DC; December 4-9, 1993.

US Army. Permethrin exposure criteria. Memo (with attachments) from Col. Lyman W. Roberts, Office of the Surgeon General, to Dr. Kulbir Bakshi, National Research Council, Washington, D.C.; March 31, 1993.

US Army. *Quantitative risk assessment, permethrin-impregnated battle dress uniform.* Transmitted by U.S. Army Medical Materiel Development Activity under cover entitled Insect/Arthropod Repellent Protective Treatment for Military Battle Dress Uniform. EPA Reg. No. Basic Formulation FE C75, Vol. 3, Permethrin Quantitative Risk Assessment, Registration Supplement, Dec. 8, 1989.

US Army Environmental Hygiene Agency. Field Evaluation of a Permethrin-Impregnated Battle Dress Uniform (BDU) Versus a Permethrin-Sprayed BDU for Prevention of Tick Bite. USAEHA Special Study No. 16 44 0600-89. Edgewood, Md: U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground; 1988.

US Army Natick Research, Development, and Engineering Center. *August Progress Report of Arthropod Repellent Impregnant Project (No. Washes Vs. Mosquito Efficacy).* U.S. Army Natick Research, Development, and Engineering Center; 1987.

van den Bercken J, Kroese ABA, Akkermans LM. 1979. Effects of insecticides on the sensory nervous system. In: T Narahashi, ed. *Neurotoxicology of Insecticides and Pheromones.* New York: Plenum Press; 1979:183-210.

van der Rhee HJ, Farquhar JA, Vermeulen NP. Efficacy and transdermal absorption of permethrin in scabies patients. *Acta Derm Venereol.* 1989;69:170-173.

Vijverberg HPM, van den Berchen J. Neurotoxicological effects and the mode of action of pyrethroid insecticides. *Crit Rev Toxicol.* 1990;21:105-126.

Wallwork LM, Clampitt RB, Malone JC. *10-Day Cumulative Oral Toxicity Study With 21Z73 in Mice.* HEFG 74-9. Wellcome Research Laboratories; 1974.

Wallwork LM, Clampitt RB, Malone JC. *10-Day Cumulative Oral Toxicity Study With 21Z73 in Rats.* HEFG 74-10. Wellcome Research Laboratories; 1974.

Wallwork LM, Poll GS, Malone JC. *Effect on the Rat Oral Toxicity of Changes in the Cis/Trans With 21Z73 (NRDC 143) Series.* HEFG 75-5. Wellcome Research Laboratories; 1975.

Woodruff RC, Phillips JP, Irwin D. Pesticide-induced complete and partial chromosome loss in screens with repair-defective females of *Drosophila melanogaster. Environ Mutagen.* 1983;5:835-846.

World MJ. Pestilence, war, and lice. *Lancet.* 1993;342(888):1192.

Reynolds J, Piercy DWT, Clampitt RB, et al. *Permethrin Oral Administration to Dogs for 6 Months.* HEFG 78-14. Wellcome Research Laboratories; 1978.

Richards D, Banham PB, Chart IS, et al. *PP557: Two-Year Feeding Study in Rats.* CTL/P/357. Imperial Chemical Industries Ltd; 1977.

Richards D, Banham PB, Kilmartin M, Weight TM. *Permethrin: Teratogenicity Study in the Rabbit.* CTL/P/523. Imperial Chemical Industries Ltd; 1980

Rishikesh N, Clarke JL, Martin HL, King JS, Pearson J. *Evaluation of Decamethrins and Permethrin Against Anopheles gambiae and Anopheles funestus in a Village Trial in Nigeria.* WHO/VBC/78.679. Division of Vector Biological Control, World Health Organization; 1978.

Robbins PJ, Cherniack MG. Review of the biodistribution and toxicity of the insect repellant *N, N*-Diethyl-*m*-toluamide (DEET). *J Toxicol Environ Health.* 1986;18:503-525.

Robinson P. *Permethrin: Acute Dermal Toxicity to the Rat.* CTL/P/2454. Imperial Chemical Industries Ltd; 1989.

Robinson P. *Permethrin: Skin Irritation to the Rabbit.* Rep. No. CTL/P/2452. Imperial Chemical Industries Ltd; 1989.

Ross DB, Prentice DE. *Examination of Permethrin (PP557) for Neurotoxicity in the Domestic Hen.* ICI/157-NT/77468. Huntington Research Centre, Imperial Chemical Industries Ltd; 1977.

Ruzo LO, Casida JE. Metabolism and toxicology of pyrethroids with dihalovinyl substituents. *Environ Health Perspect.* 1977;21:285-292.

Sasinovich LM, Panshina TN. Substantiation of hygienic rules for synthetic pyrethroid content in the work zone air. *Gig Tr Prof Zabol.* 1987;8:48-50.

Schreck CE, Carlson DA, Weidhaas DE, Posey K , Smith D. Wear and aging tests with permethrin-treated cotton-polyester fabric. *J Econ Entomol.* 1980;73:451-453.

Schreck CE, Haile DG, Kline DL. The effectiveness of permethrin and DEET, alone or in combination, for protection against *Aedes taeniorhynchus. Am J Trop Med Hyg.* 1984;33:725-730.

Schreck CE, Kane F, Carlson DA. Permethrin impregnations of military fabrics: an evaluation of application rates and industrial methods by bioassay and gas chromatographic analysis. *Soap Cosmetics Chem Spec.* (Aug.); 1982.

Schreck CE, Kline DL. Personal protection afforded by controlled-release topical repellents and permethrin-treated clothing against natural populations of *Aedes taeniorhynchus. J Am Mosq Control Assoc.* 1989;5:77-80.

Schreck CE, McGovern TP. Repellents and other personal protection strategies against *Aedes albopictus. J Am Mosq Control Assoc.* 1989;5:247-250.

Schreck CE, Mount GA, Carlson DA. Pressurized sprays of permethrin on clothing for personal protection against the lone star tick (Acari: Ixodidae). *J Econ Entomol.* 1982;75:1059-1061.

Schreck CE, Mount GA, Carlson DA. Wear and wash persistence of permethrin used as a clothing treatment for personal protection against the lone star tick (Acari: Ixodidae). *J Med Entomol.* 1982;19:143-146.

Schreck CE, Posey K, Smith D. Durability of permethrin as a potential clothing treatment to protect against blood-feeding arthropods. *J Econ Entomol.* 1978;71:397-400.

Schreck CE, Snoddy EL, Mount GA. Permethrin and repellents as clothing impregnants for protection from the Lone Star Tick. *J Econ Entomol.* 1980;73:436-439.

Schreck CE, Snoddy EL, Spielman A. Pressurized sprays of permethrin or DEET on military clothing for personal protection against *Ixodes dammini* (Acari: Ixodidae). *J Med Entomol.* 1986;23:396-399.

Schultz MW, Gomez M, Hansen RC, et al. Comparative study of 5% permethrin cream and 1% lindane lotion for the treatment of scabies. *Arch Dermatol.* 1990;126:167-170.

Shah PV, Fisher HL, Sumler MR, Monroe RJ. Comparison of the penetration of 14 pesticides through the skin of young and adult rats. *J Toxicol Environ Health.* 1987;21:353-366.

Shah PV, Monroe RI, Guthrie FE. Comparative rates of dermal penetration of insecticides in mice. *Toxicol Appl Pharmacol.* 1981;59:414-423.

Shapiro R. *Permanone Multi-Use Insecticide Spray: EPA Acute Dermal Toxicity Limit Test.* Product Safety Labs, Inc; 1989.

Yonkosky D, Ladia L, Gackenheimer L, Schultz MW. Scabies in nursing homes: an eradication program with permethrin 5% cream. *J Am Acad Dermatol* 1990;23:1133-1136.

LEISHMANIASIS

Abdel-Aal H, Morsy TA, Hawwary GH. Clinical forms of cutaneous leishmaniasis in Riyadh, Saudi Arabia. *JPMA J Pak Med Assoc.* 1975;25(9):239-242.

Abdel Wahab RM, el Garem AA, Morsy TA, Essa MH. The histopathological picture of cutaneous leishmaniasis in western part of Saudi Arabia. *J Egypt Soc Parasitol.* 1985;15(2):381-386.

Abdel Wahab RM, Morsy TA, Essa MH. Clinical and laboratory aspects of visceral leishmaniasis in Gizan, Saudi Arabia. *J Egypt Soc Parasitol.* 1984;14(2):563-572.

Abdel Wahab RM, Morsy TA, el Garem AA, Bahgat A, Essa MH. Introduced cases of cutaneous leishmaniasis in Egypt. *J Egypt Soc Parasitol.* 1986;16(1):9-16.

Akuffo H, Darce M, Maasho K, Berhan TY. *In vivo* evaluation of immune responses in Leishmaniasis: the use of cross-species leishmanin preparations for skin testing. *Am J Trop Med Hyg.* 1995;53(1):16-22.

Albanese G, Giorgetti P, Santagostino L, Crippa D, Sala G. Cutaneous leishmaniasis. *Arch Dermatol.* 1989;125:1540-1542.

Alexander J, Russell DG. Parasite antigens, their role in protection, diagnosis and escape: the leishmaniases. *Curr Top Microbiol Immunol.* 1985;120:43-67.

al-Fouzan AS, al Saleh QA, Najem NM, Rostom AI. Cutaneous leishmaniasis in Kuwait. Clinical experience with itraconazole. *Int J Dermatol.* 1991;30(7):519-521.

Al-Gindan Y, Abdul-Aziz O, Kubba R. Cutaneous leishmaniasis in Al-Hassa, Saudi Arabia. *Int J Dermatol.* 1984;23:194-197.

al-Jurayyan NA, al Ayed IH, al-Nasser MN, al-Mugeiren MM, Boohene AG, al Herbish AS. Visceral leishmaniasis in infancy and childhood epidemiology and clinicopathological study of 63 cases in Al-Baha Province, Saudi Arabia. *J Trop Pediatr.* 1992;38(1):12-16.

al-Shammari SA, Khoja TA, Fehr A. Cutaneous leishmaniasis in Riyadh region: four-year study of the epidemiologic and clinical features. *Int J Dermatol.* 1992;31(8):565-567.

al-Taqi M, Behbehani K. Cutaneous leishmaniasis in Kuwait. *Ann Trop Med Parasitol.* 1980;74:495-501.

al-Zahrani MA, Peters W, Evans DA, Chin C, Smith V, Lane RP. *Phlebotomus sergenti*, a vector of *Leishmania tropica* in Saudi Arabia. *Trans R Soc Trop Med Hyg.* 1988;82:416.

al-Zahrani MA, Peters W, Evans DA, Smith V, Ching CI. *Leishmania* infecting man and wild animals in Saudi Arabia. 5. Diversity of parasites causing visceral leishmaniasis in man and dogs in the south-west. *Trans R Soc Trop Med Hyg.* 1989;83:503-509.

al-Zahrani MA, Peters W, Evans DA, Smith V, Ching Chin I. *Leishmania* infecting man and wild animals in Saudi Arabia. 6. Cutaneous leishmania of man in the south-west. *Trans R Soc Trop Med Hyg.* 1989;83:621-628.

al-Zahrani MA, Peters W, Evans DA. Visceral leishmaniasis in man and dogs in south-west Saudi Arabia. *Trans R Soc Trop Med Hyg.* 1988;82(6):857.

Alimohammadian MH, Kivanjah M, Pak F, Gaznavia A, Kharazmi A. Evaluation of the efficacy of Iran leishmanin and comparison with leishmanins from Wellcome (UK) and Roma (Italy) in cured cutaneous leishmaniasis patients. *Trans R Soc Trop Med Hyg.* 1993;87(5):550-551.

Amichai B, Finkelstein E, Grunwald MH, Halevy S. Think cutaneous leishmaniasis. *Aust Fam Physician.* 1993;22(7):1213-1214, 1217.

Ankney B. Unusual leishmaniasis found in troops. *US Med.* 1991;23/24:1, 26-27.

Ashford RW, Bettini S. Ecology and epidemiology. In: Peters W, Killick-Kendrick R, eds. *The Leishmaniasis in Biology and Medicine.* Vol. 1. London, England: Academic Press; 1987:365-424.

Badaro R, Falcoff E, Badaro FS, et al. Treatment of visceral leishmaniasis with pentavalent antimony and interferon gamma. *N Engl J Med.* 1990;322:16-21.

Leishmaniasis

Badaro R, Jones TC, Carvalho EM, et al. New perspectives on a subclinical form of visceral leishmaniasis. *J Infect Dis.* 1986;154:1003-1011.

Ballou RW, Ohl CA. *Viscerotropic Leishmaniasis in Returnees From Operation Desert Shield/Storm.* Chester, MD: Armed Forces Epidemiological Board, Parson's Island;1992.

Belazzoug S. Leishmaniasis in Mediterranean countries. *Vet Parasitol.* 1992;44(1-2):15-19.

Berman JD. Chemotherapy for leishmaniasis: biochemical mechanisms, clinical efficacy, and future strategies. *Rev Infect Dis.* 1988;10:560-586.

Bienzle U, Ebert F, Dietrich M. Cutaneous leishmaniasis in Eastern Saudi Arabia: epidemiological and clinical features in a nonimmune population living in an endemic area. *Tropenmed Parasitol.* 1978;29:188-193.

Blackwell JM. Leishmaniasis epidemiology: all down to the DNA. *Parasitology.* 1992;104 (suppl):S19-S34.

Bogdan C, Stosiek N, Fuchs H, et al. Detection of potentially diagnostic leishmanial antigens by Western blot analysis of sera from patients with kala-azar or multilesional cutaneous leishmaniasis [letter]. *J Infect Dis.* 1990;162(6): 1417-1418.

Bouree P, Belec L. Leishmaniasis: report of 33 cases and a review of the literature [editorial]. *Comp Immunol Microbiol Infect Dis.* 1993;16(4):251-265.

Boyer MH. Visceral leishmaniasis in Desert Storm veterans [letter]. *N Engl J Med.* 1993;329(20):1503-1504.

Bryceson ADM. Leishmaniasis. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford Textbook of Medicine. 2nd ed. Vol. 1.* Oxford, England: Oxford University Press; 1987:5.524-5.532.

Centers for Disease Control. Viscerotropic leishmaniasis in persons returning from Operation Desert Storm-1990-1991. *JAMA.* 1992;267(11):1444-1446.

Conti R, Parenti F. Rifampin therapy for brucellosis, flavobacterium meningitis, and cutaneous leishmaniasis. *Rev Infect Dis.* 1983;5(suppl 3):S600-S605.

Cotton P. Gulf War symptoms remain puzzling [news]. *JAMA.* 1992;268(19):2619.

Defense Pest Management Information Analysis Center. *Leishmaniasis: Review of Desert Storm Cases.* Technical Information Bulletin. Washington, DC: Defense Pest Management Information Analysis Center; 1992;July/August:8.

de Letona JML, Vazquez CM, Maestu RP. Visceral leishmaniasis as an opportunistic infection. *Lancet.* 1986;1:1094.

Dillon DC, Day CII, Whittle JA, Magill AJ, Reed SG. Characterization of a *Leishmania tropica* antigen that detects immune responses in Desert Storm viscerotropic leishmaniasis patients. *Proc Nat Acad Sci USA.* 1995;92:7981-7985.

Dye C. Leishmaniasis epidemiology: the theory catches up. *Parasitology.* 1992;104(suppl):S7-S18.

Edrissian GH, Mohammadi M, Kamani A, Afshar A, Hafezi R, Ghorbani M, Gharagozloo AR. Bacterial infections in suspected cutaneous leishmaniasis lesions. *Bull World Health Organ.* 1990;68(4):473-477.

Elbihari S, Kawasmeh ZA, Al-Naiem A. Al-Atiya S. *Leishmania* infecting man and wild animals in Saudi Arabia 3. Leishmaniasis in *Psammomys obesus* Cretzschmar in Al-Ahsa oasis. *Trop Med Parasitol.* 1987;38:89-92.

el-Darouti MA, Al-Rubaie SM. Cutaneous leishmaniasis. Treatment with combined cryotherapy and intralesional stibogluconate injection. *Int J Dermatol.* 1990;29:56-59.

el-Safi SH, Peters W. Studies on the leishmaniases in the Sudan. 1. Epidemic of cutaneous leishmaniasis in Khartoum. *Trans R Soc Trop Med Hyg.* 1991;85(1):44-47.

el-Safi SH, Peters W, el-Toam B, el-Kadarow A, Evans DA. Studies on the leishmaniases in the Sudan. 2. Clinical and parasitological studies on cutaneous leishmaniasis. *Trans R Soc Trop Med Hyg.* 1991;85(4):457-464.

el-Safi SH, Peters W, Evans DA. Studies on the leishmaniases in the Sudan. 3. Clinical and parasitological studies on visceral and mucosal leishmaniasis. *Trans R Soc Trop Med Hyg.* 1991;85(4):465-470.

el-Sibae MM, Eesa NM. A study on *Phlebotomus* species, the vectors of leishmaniasis in Gassim, Saudi Arabia. *J Egypt Soc Parasitol.* 1993;23(1):231-238.

Leishmaniasis

Fernandez-Guerrero ML, Aguado J, Buzon L, et al. Visceral leishmaniasis in immunocompromised hosts. *Am J Med.* 1987;83:1098-1102.

Fryauff DJ, Modi GB, Mansour NS, Kreutzer RD, Soliman S, Youssef FG. Epidemiology of cutaneous leishmaniasis at a focus monitored by the multinational force and observers in the northeastern Sinai Desert of Egypt. *Am J Trop Med Hyg.* 1993;49(5):598-607.

Funke BJ. Leishmaniasis update [letter]. *Postgrad Med.* 1992;91(4):79.

Giladi M, Block C, Danon YL, Schinder E, Greenblatt CL. Local environmental risk factors in the acquisition of cutaneous leishmaniasis. *Isr J Med Sci.* 1988;24(3):185-187.

Giladi M, Danon YL, Greenblatt C, Schinder E, Nili E. Keziot-a new endemic site of cutaneous leishmaniasis in Israel. An epidemiological and clinical study of a non-immune population entering an endemic area. *Trop Geogr Med.* 1985;37(4):298-303.

Greenblatt CL, Schnur LF. Leishmaniasis recently imported into Israel. *Isr J Med Sci.* 1985;21(8):709-710.

Griffiths WAD. Old world cutaneous leishmaniasis. In: Peters W, Killick-Kendrick R, eds. *The Leishmaniases in Biology and Medicine. Vol 2.* London: Academic Press; 1987:617-636.

Grimaldi G Jr, McMahan-Pratt D. Leishmaniasis and its etiologic agents in the New World: an overview. *Prog Clin Parasitol.* 1991;2:73-118.

Grimaldi G Jr, Tesh RB. Leishmaniases of the New World: current concepts and implications for future research. *Clin Microbiol Rev.* 1993;6(3):230-250.

Grimaldi G Jr, Tesh RB, McMahon-Pratt D. A review of the geographic distribution and epidemiology of leishmaniasis in the New World. *Am J Trop Med Hyg.* 1989;41(6):687-725.

Grogl M, Daugirda JL, Hoover DL, Magill AJ, Berman JD. Survivability and infectivity of viscerotropic *Leishmania tropica* from Operation Desert Storm participants in human blood products maintained under blood bank conditions. *Am J Trop Med Hyg.* 1993;49(3):308-315.

Grogl M, Oduola AM, Cordero LD, Kyle DE. *Leishmania* spp.: Development of pentostam-resistant clones in vitro by discontinuous drug exposure. *Exp Parasitol.* 1989;69:78-90.

Grogl M, Thomason TN, Franke ED. Drug resistance in leishmaniasis: its implication in systemic chemotherapy of cutaneous and mucocutaneous disease. *Am J Trop Med Hyg.* 1992;47:117-126.

Gunby P. Desert Storm veterans now may donate blood; others call for discussion of donor tests [news]. *JAMA.* 1993;269(4):451-452.

Hepburn NC, Tidman MJ, Hunter JA. Cutaneous leishmaniasis in British troops from Belize. *Br J Dermatol.* 1993;128(1):63-68.

Herwaldt BL, Berman JD. Recommendations for treating leishmaniasis with sodium stibogluconate (Pentostam) and review of pertinent clinical studies. *Am J Trop Med Hyg.* 1992;46:296-306.

Hussien MS, Behbehani K. The epidemiology of leishmaniasis in Kuwait, 1. The occurrence and distribution of *Phlebotomus* sandflies (*Diptera, Psuchodidae*). *Zeitschrift fur Angewandte Entomologie.* 1976;81:433-440.

Ibrahim EA, al-Zahrani MA, al-Tuwaigri AS, al-Shammary FJ, Evans DA. Leishmania infecting man and wild animals in Saudi Arabia. 9. The black rat (Rattus rattus) a probable reservoir of visceral leishmaniasis in Gizan province, south-west Saudi Arabia. *Trans R Soc Trop Med Hyg.* 1992;86(5):513-514.

Kalra NL, Bang YH. Sandfly vector competence for transmission of *Leishmania* parasite in tropical Asia. *J Commun Dis.* 1986;18(1):28-34.

Khairy N, el-Hashimi W. A study of three isolates of *Leishmania donovani* from patients with kala-azar in Iraq. *Ann Trop Med Parasitol.* 1980;74:29-35.

Kibbi AG, Karam PG, Kurban AK. Sporotrichoid leishmaniasis in patients from Saudi Arabia: clinical and histologic features. *J Am Acad Dermatol.* 1987;17:759-764.

Killick-Kendrick R, Leaney AJ, Peters W, Rioux J-A, Bray RS. Zoonotic cutaneous leishmaniasis in Saudi Arabia: the incrimination of *Phlebotomus papatasi* as the vector in the Al-Hassa oasis. *Trans R Soc Trop Med Hyg.* 1985;79:252-255.

Killick-Kendrick R, Peters W. Leishmaniasis and 'Desert Storm' [letter]. *Trans R Soc Trop Med Hyg.* 1992;86(6):698.

Kreutzer RD, Grogl M, Neva FA, Fryauff DJ, Magill AJ, Aleman-Munoz MM. Identification and genetic comparison of leishmanial parasites causing viscerotropic and cutaneous disease in soldiers returning from Operation Desert Storm. *Am J Trop Med Hyg.* 1993;49:357-363.

Leishmaniasis

Kreutzer RD, Souraty N, Semko ME. Biochemical identities and differences among *Leishmania* species and subspecies. *Am J Trop Med Hyg.* 1987;36:22-32.

Kreutzer RD, Christensen HA. Characterization of *Leishmania* spp. by isozyme electrophoresis. *Am J Trop Med Hyg.* 1980;29:199-208.

Kubba R, El-Hassan AM, Al-Gindan Y, Orner AHS, Kutty MK, Saeed MBM. Dissemination in cutaneous leishmaniasis. *Int J Dermatol.* 1987;26:300-304.

Le Blancq SM, Peters W. Leishmania in the Old World: 2. Heterogeneity among *L. tropica* zymodemes. *Trans R Soc Trop Med Hyg.* 1986;80:113-119.

Leishmaniasis and the Arabian Gulf. *Commun Dis Rep CDR Wkly.* 1992;2(14):61.

Lemesre JL, Darcy F, Kweider M, Capron A, Santoro F. Requirements of defined cultivation conditions for standard growth of *Leishmania* promastigotes in vitro. *Acta Trop (Basel).* 1988;45:99-108.

Lesho EP. Leishmaniasis. Another threat to Persian Gulf veterans. *Postgrad Med* 1991;90(8):213-214, 216-217.

Magill AJ, Gasser RA Jr, Oster CN, Grogl M, Sun W. Viscerotropic leishmaniasis in persons returning from Operation Desert Storm 1990-1991. *Arch Dermatol* 1992;128(8):1033-1034.

Magill AJ, Grogl M, Gasser RA, Oster CN. Visceral leishmaniases in Desert Storm veterans [letter]. *N Engl J Med.* 1993;329:1504.

Magill AJ, Grogl M, Gasser RA, Sun W, Oster CN. Visceral infection caused by *Leishmania tropica* in veterans of Operation Desert Storm. *N Engl J Med.* 1993;328:1383-1387.

Magill AJ, Grogl M, Johnson SC, Gasser RA. Visceral infection due to *Leishmania tropica* in a veteran of Operation Desert Storm who presented 2 years after leaving Saudi Arabia [letter]. *Clin Infect Dis.* 1994;19:805-806.

Mahmoud AA, Bolbol AS, Sholou YA. Some aspects of the epidemiology of cutaneous leishmaniasis in the Al-Kharaj area of Saudi Arabia. *Ann Trop Med Parasitol.* 1984;78:605-609.

Mansour NS, Pryaufff DJ, Modi GB, Mikhail EM, Youssef FG. Isolation and characterization of *Leishmania major* from *Phlebotomus papatasi* and military personnel in north Sinai, Egypt. *Trans R Soc Trop Med Hyg.* 1991;85(5):590-591.

Mebrahto Y, Lawyer P, Githure J, et al. Visceral leishmaniasis unresponsive to pentostam caused by *Leishmania tropica* in Kenya. *Am J Trop Med Hyg.* 1989;41:289-294.

Melby PC, Kreutzer RD, McMahon-Pratt D, Gam AA, Neva FA. Cutaneous leishmaniasis: review of 59 cases seen at the National Institutes of Health. *Clin Infect Dis.* 1992;15(6):924-937.

Meleney HE. Leishmaniasis. In: Hoff EC, ed. *Preventive Medicine in World War II. Vol. 7. Communicable Diseases.* Washington, DC: Office of the Surgeon General, U.S. Department of the Army; 1964:73-77.

MMWR (Morbidity and Mortality Weekly Report). Viscerotropic leishmaniasis in persons returning from Operation Desert Storm-1990-1991. *MMWR.* 1992;41:131-134.

Mogul A, Grog M, Ganser RA, Sun W. Osier CAN. Visceral infection caused by *Leishmania tropica* in veterans of Operation Desert Storm. *N Engl J Med.* 1993;328:1383-1387.

Most H, Lavietes PH. Kala-azar in American military personnel: report of 30 cases. *Medicine* (Baltimore). 1947;26:221-284.

Nascimento E, Mayrink W, da Costa CA, et al. Vaccination of humans against cutaneous leishmaniasis: cellular and humoral immune responses. *Infect Immun.* 1990;58(7):2198-2203.

Neal RA, Reeves A, Peters W. *Leishmania* infecting man and wild animals in Saudi Arabia. 7. Patrial protection of mice against *Leishmania major* by prior infection with *L. arabica. Trans R Soc Trop Med Hyg.* 1990;84(2): 233-238.

Norton SA, Frankenburg S, Klaus SN. Cutaneous leishmaniasis acquired during military service in the Middle East. *Arch Dermatol.* 1992;128(1):83-87.

Ohl CA, Hyams KC, Malone JD, Oldfield E 3d. Leishmaniasis among Desert Storm veterans: a diagnostic and therapeutic dilemma. *Mil Med.* 1993;158(11):726-729.

Oren R, Schnur LF, Yehuda DB, Mayner V, Okon E. Rachmilewitz EA. Visceral leishmaniasis: a difficult diagnosis and unusual causative agent. *J Infect Dis.* 1991;164:746-749.

Oster CN, Chulay JD. Visceral leishmaniasis (kala-azar). In: Strickland GT, ed. *Hunter's Tropical Medicine.* 7th ed. Philadelphia, Pa: W.B. Saunders; 1991:642-648.

Oster CN, Sanford JP. Febrile illness in a Desert Storm veteran. *Hosp Pract (Off Ed).* 1992;27(11):145-148, 151, 155-160.

Palma G, Gutierrez Y. Laboratory diagnosis of *Leishmania. Clin Lab Med.* 1991;11(4):909-922.

Pearson RD, De Queiroz Sousa A. *Leishmania* species: visceral (kala-azar), cutaneous, and mucosal leishmaniasis. In: Mandell GL, Douglas G Jr, Bennett JE, eds. *Principles and Practice of Infectious Diseases.* 3rd ed. New York: Longman (Churchill Livingstone); 1990:2066-2077.

Perich MJ, Hoch AL, Rizzo N, Rowton ED. Insecticide barrier spraying for the control of sand fly vectors of cutaneous leishmaniasis in rural Guatemala. *Am J Trop Med Hyg.* 1995;52(6):485-488.

Peters W, Al-Zahrani MA. The leishmaniases-a public health problem in Saudi Arabia. *Saudi Med J.* 1987;8:333-343.

Rashti MA, Panah HY, Mohamadi HS, Jedari M. Susceptibility of *Phlebotomus papatasi* (Diptera: Psychodidae) to DDT in some foci of cutaneous leishmaniasis in Iran. *J Am Mosq Control Assoc.* 1992;8(1):99-100.

Saenz RE, Paz H, Berman JD. Efficacy of ketoconazole against *Leishmania braziliensis panamensis* cutaneous leishmaniasis. *Am J Med.* 1990;89:147-155.

Saf'janova VM. Phlebotominae sandflies as the key link of *Leishmania* parasitic systems. *Parassitologia.* 1991;33(suppl):505-511.

Sanchez JL, Diniega BM, Small JW, et al. Epidemiologic investigation of an outbreak of cutaneous leishmaniasis in a defined geographic focus of transmission. *Am J Trop Med Hyg.* 1992;47(1):47-54.

Schnur LF, Chance ML, Ebert F, Thomas SC, Peters W. The biochemical and serological taxonomy of visceralizing *Leishmania. Ann Trop Med Parasitol.* 1981;75:131-144.

Selim MM, Vlasin Z, Jaroskova L. Leishmaniasis. Currently recommended treatment. *Int J Dermatol.* 1990;29:318-321.

Symmers WSTC. Leishmaniasis acquired by contagion: a case of marital infection in Britain. *Lancet.* 1960;1:127-132.

Tarizzo ML, Bracken HA, Strait DJ. A case of visceral leishmaniasis in Saudi Arabia. *Am J Trop Med Hyg.* 1953;2:846-849.

Wali JP, Aggarwal P, Gupta U, et al. Ketoconazole in the treatment of antimony and pentamidine-resistant kala azar. *J Infect Dis.* 1992;166:215-216.

Walton BC, Brooks WH, Arjona I. Serodiagnosis of American leishmaniasis by indirect fluorescent antibody test. *Am J Trop Med Hyg.* 1972;21:296-299.

Weinrauch L, Livshin R, el-On 1. Ketoconazole in cutaneous leishmaniasis. *Br J Dermatol.* 1987;17:666-668.

MULTIPLE CHEMICAL SENSITIVITY

Adler T. Desert Storm's medical quandary: Do Iraqi chemical and biological agents explain Gulf War syndrome? *Sci News.* 1994;145:394-395.

Advancing the understanding of multiple chemical sensitivity. Proceedings of the Association of Occupational and Environmental Clinics Workshop on Multiple Chemical Sensitivity. Washington, DC, 20-21 September 1991. *Toxicol Ind Health.* 1992;8(4):1-257.

Albright JF, Goldstein RA. Is there evidence of an immunologic basis for multiple chemical sensitivity? *Toxicol Ind Health.* 1992;8(4):215-219.

Bascom R. Multiple chemical sensitivity: a respiratory disorder? *Toxicol Ind Health.* 1992;8(4):221-228.

Bell IR, Miller CS, Schwartz GE. An olfactory-limbic model of multiple chemical sensitivity syndrome: possible relationships to kindling and affective spectrum disorders. *Biol Psychiatry.* 1992;32(3):218-242.

Bell IR, Schwartz GE, Peterson JM, Amend D. Self-reported illness from chemical odors in young adults without clinical syndromes or occupational exposures. *Arch Environ Health.* 1993;48(1):6-13.

Black DW. Environmental illness and misdiagnosis-a growing problem. *Regul Toxicol Pharmacol.* 1993;18(1):23-31.

Multiple Chemical Sensitivity

Black DW, Rathe A, Goldstein RB. Measures of distress in 26 environmentally ill subjects. *Psychosomatics.* 1993;34(2):131-138.

Blanck RR. *Trip Report of Meetings With Coalition Members of Operation Desert Storm/Desert Shield Concerning Reports of Low Level Chemical Warfare Agent Detection and Health of Their Forces.* Falls Church, VA: Dept of the Army, Office of the Surgeon General; 1994.

Casanova-Roig R. Clinical ecology, multiple chemical sensitivity (MCS): the debate [letter]. *Bol Asoc Med P R.* 1991;83(12):553-556.

Cullen MR. The worker with multiple chemical sensitivities: an overview. *Occup Med.* 1987;2(4):655-661.

Davidoff LL, Callender TJ, Morrow LA, Ziem G. Multiple chemical sensitivities (MCS) [letter]. *J Psychosom Res.* 1991;35(4-5):621-623.

Gots RE, Hamosh TD, Flamm WG, Carr CJ. Multiple chemical sensitivities: a symposium on the state of the science. *Regul Toxicol Pharmacol.* 1993;18(1):61-78.

Jewett DL. Research strategies for investigating multiple chemical sensitivity. *Toxicol Ind Health.* 1992;8(4):175-179.

Kilburn KH. Symptoms, syndrome, and semantics: multiple chemical sensitivity and chronic fatigue syndrome [editorial]. *Arch Environ Health.* 1993;48(5):368-369.

Levin AS, Byers VS. Multiple chemical sensitivities: a practicing clinician's point of view. Clinical and immunologic research findings. *Toxicol Ind Health.* 1992;8(4):95-109.

Miller CS. Possible models for multiple chemical sensitivity: conceptual issues and role of the limbic system. *Toxicol Ind Health.* 1992;8(4):181-202.

Nethercott JR, Davidoff LL, Curbow B, Abbey H. Multiple chemical sensitivities syndrome: toward a working case definition. *Arch Environ Health.* 1993;48(1):19-26.

Rest KM. Advancing the understanding of multiple chemical sensitivity (MCS): overview and recommendations from an AOEC workshop. *Toxicol Ind Health.* 1992;8(4):1-13.

Richter E. Multiple chemical sensitivity: respect the observations ... suspect the interpretations? [editorial]. *Arch Environ Health.* 1993;48(5):366-367.

Simon GE. Epidemic multiple chemical sensitivity in an industrial setting. *Toxicol Ind Health.* 1992;8(4):41-46.

Simon GE. Psychiatric treatments in multiple chemical sensitivity. *Toxicol Ind Health.* 1992;8(4):67-72.

Simon GE, Daniell W, Stockbridge H, Claypoole K, Rosenstock L. Immunologic, psychological, and neuropsychological factors in multiple chemical sensitivity. A controlled study. *Ann Intern Med.* 1993;119(2):97-103.

Smith WJ, Dunn MA. Medical defense against blistering chemical warfare agents. *Arch Dermatol.* 1991;127(8):1207-1213.

Staudenmayer H, Selner JC, Buhr MP. Double-blind provocation chamber challenges in 20 patients presenting with multiple chemical sensitivity. *Regul Toxicol Pharmacol.* 1993;18(1):44-53.

U.S. chemical and biological warfare-related dual use exports to Iraq and their possible impact on the health consequences of the Persian Gulf war. A report of Chairman Donald W. Riegle, Jr. and Ranking Member Alfonse M. D'Amato of the Committee on Banking, Housing and Urban Affairs with Respect to Export Administration. Washington, DC: United States Senate; May 25, 1994.

Waddell WJ. The science of toxicology and its relevance to MCS. *Regul Toxicol Pharmacol.* 1993;18(1):13-22.

Watson AP, Griffin GD. Toxicity of vesicant agents scheduled for destruction by the Chemical Stockpile Disposal Program. *Environ Health Perspect.* 1992;98:259-280.

Welch LS, Sokas R. Development of multiple chemical sensitivity after an outbreak of sick-building syndrome. *Toxicol Ind Health.* 1992;8(4):47-50.

Ziem GE. Multiple chemical sensitivity: treatment and follow-up with avoidance and control of chemical exposures. *Toxicol Ind Health.* 1992;8(4):73-86.

Other Infectious Disease

David JR, Butterworth AE. Immunity to schistosomiasis: advances and prospects. *Ann Intern Med.* 1979;91:641-643.

DeFraites RF, Wanat ER, Norwood AE, Williams S, Cowan D, Callahan T. *Investigation of a Suspected Outbreak of an Unknown Disease Among Veterans of Operation Desert Shield/Storm, 123rd Army Reserve Command, Fort Benjamin Harrison, Indiana, April 1992.* Washington, DC: Epidemiology Consultant Service, Division of Preventive Medicine, Walter Reed Army Institute of Research; 1992.

Deller JJ Jr, Russell PK. An analysis of fevers of unknown origin in American soldiers in Vietnam. *Ann Intern Med.* 1967;66:1129-1143.

Division of Control of Tropical Diseases. World malaria situation, 1988. *World Health Stat Q.* 1990;43:68-78.

Eitrem R, Vene S, Niklasson B . Incidence of sandfly fever among Swedish United Nations soldiers during 1985. *Am J Trop Med Hyg.* 1990;43:207-211.

Elhag KM, Mustafa AK, Sethi SK. Septicaemia in a teaching hospital in Kuwait-I: incidence and aetiology. *J Infect.* 1985;10:17-24.

el-Hazmi MA. Hepatitis B virus in Saudi Arabia. *J Trop Med Hyg.* 1989;92:56-51.

el-Hazmi MA. Hepatitis A antibodies: prevalence in Saudi Arabia. *J Trop Med Hyg.* 1989;92:427-430.

Gasser RA Jr, Magill AJ, Oster CN, Tramont EC. The threat of infectious disease in Americans returning from Operation Desert Storm. *N Engl J Med.* 1991;324(12):859-864.

Gordon JE. General considerations of modes of transmission. In: Hoff EC, ed. *Preventive Medicine in World War II. Vol. 4 Communicable Diseases.* Washington, DC: Office of the Surgeon General, U.S. Department of the Army; 1958:8-38.

Gremillion DH, Geckler RW, Kuntz RE, Marraro RV. Schistosomiasis in Saudi Arabian recruits. A morbidity study based on quantitative egg excretion. *Am J Trop Med Hyg.* 1978;27:924-927.

Gross EM, Arbeli Y, Bearman JE, et al. Spotted fever and murine typhus in the Negev desert region of Israel, 1981. *Bull World Health Organ.* 1984;62:301-306.

Henig M, Sabin AB. Sandfly fever (*Pappataci, Phlebotomus*, three day fever). In: Coates JB Jr, Hoff EC, Hoff PM, eds. *Communicable Diseases: Arthropodborne Diseases Other Than Malaria. Vol. 7 of Preventive Medicine in World War II.* Washington, DC: Government Printing Office, 1964:109-174.

Hepburn NC, Brooks TJG. An outbreak of chickenpox in a military field hospital-the implications for biological warfare. *J R Soc Med.* 1991;84:721-722.

Heppner DG, Magill AJ, Gasser RA, Oster CN. The threat of infectious diseases in Somalia. *N Engl J Med.* 1993;328:1061-1068.

Herron A, Dettleff G, Hixon B, et al. Influenza vaccination in patients with rheumatic diseases: safety and efficacy. *JAMA.* 1979;242:53-56.

Hertig M, Sabin AB. Sandfly fever (*pappataci, Phlebotomus*, three-day fever). In: Hoff EC, ed. *Preventive Medicine in World War II. Vol. 7: Communicable Diseases.* Washington, DC: Office of the Surgeon General, U.S. Department of the Army; 1964:109-174.

Holmes GP, Kaplan JE, Stewart JA, Hunt B, Pinsky PF, Schonberger LB. A cluster of patients with a chronic mononucleosis-like syndrome: is Epstein-Barr virus the cause? *JAMA.* 1987;257:2297-2302.

Hoogstraal H. The epidemiology of tick-borne Crimean-Congo hemorrhagic fever in Asia, Europe, and Africa. *J Med Entomol.* 1979;15:307-417.

Hornick RB. Typhoid fever. In: Evans AS, Feldman HA, eds. *Bacterial Infections in Humans: Epidemiology and Control.* New York, Ny: Plenum; 1982:659-676.

Huggins JW. Prospects for treatment of viral hemorrhagic fevers with ribavirin, a broad-spectrum antiviral drug. *Rev Infect Dis.* 1989;11(suppl 4):S750-S761.

Human plague in 1989. *Wkly Epidemiol Rec.* 1990;65:321-323.

Hussain Qadri SM, Ostrawski S, Johnson S, Flournoy DJ. Differences in antimicrobial susceptibilities of clinical isolates in Saudi Arabia and the United States. *J Natl Med Assoc.* 1987;79:433-437.

Hyams KC, Hanson K, Wignall FS, Escamilla J, Oldfield, EC. The impact of infectious diseases on the health of U.S. troops deployed to the Persian Gulf during Operations Desert Shield and Desert Storm. *Clin Infect Dis.* 1995;20:1497-1504.

OTHER INFECTIOUS DISEASE

Acute schistosomiasis with transverse myelitis in American students returning from Kenya. *MMWR*. 1984;33:445-447.

Al-Awadi AR, Al-Kazemi N, Ezzat G, et al. Murine typhus in Kuwait in 1978. *Bull World Health Organ*. 1982;60:283-289.

Al-Bustan M. Infectious and parasitic disease mortality in Kuwait and the role of health education. *Community Med*. 1988;10:197-202.

Al-Madani AA. Control of schistosomiasis in Saudi Arabia. A multidisciplinary approach. *J Egypt Soc Parasitol*. 1989;19(suppl):879-886.

Al-Madani R. Paratyphoid outbreak in children. *Journal of the Bahrain Medical Society*. 1989;1:60-63.

Al-Mofleh IA, Al-Aska AKI, Al-Nozha MM. Fever of unknown origin: experience in Riyadh, Saudi Arabia. *Annals of Saudi Medicine*. 1990;10:620-625.

Al-Nakib W, Lloyd G. El-Mekki A, Plan G, Beeson A, Southee T. Preliminary report on arbovirus-antibody prevalence among patients in Kuwait: evidence of Congo/Crimean virus infection. *Trans R Soc Trop Med Hyg*. 1984;78:474-476.

Al-Orainey IO, Saeed ES, el-Kassimi FA, al-Shareef N. Resistance to antituberculosis drugs in Riyadh, Saudi Arabia. *Tubercle*. 1989;70:207-210.

Al-Rawi TI, Thewaini AJ, Shawket AR, Ahmed GM. Skeletal brucellosis in Iraqi patients. *Ann Rheum Dis*. 1989;48:77-79.

Al-Tikriti SK, Al-Ani F, Jurji FJ, et al. Congo/Crimean haemorrhagic fever in Iraq. *Bull World Health Organ*. 1981;59:85-90

Al-Tikriti SK, Hassan FK, Moslih IM, Jurji F. Mahmud MI, Tantawi HH. Congo/Crimean haemorrhagic fever in Iraq: a seroepidemiological survey. *J Trop Med Hyg*. 1981;84:117-120.

Arfaa F. Studies on schistosomiasis in Saudi Arabia. *Am J Trop Med Hyg*. 1976;25:295-298.

Arrighi HM. Brucellosis surveillance in Saudi Arabia's Eastern Province. *Annals of Saudi Medicine*. 1986;6(suppl):S5-S10.

Ashi J, Arfaa F, Jeffri M, Suwairy M. Progress achieved in the control of schistosomiasis in Saudi Arabia. *J Trop Med Hyg*. 1989;92:27-31.

Bahar RH, Al-Suhaili AR, Mousa AM, Nawaz MK, Kaddah N, Abdel-Dayem HM. Brucellosis: appearance on skeletal imaging. *Clin Nucl Med*. 1988;13:102-106.

Bahmanyar M. Human plague episode in the district of Khawlan, Yemen. *Am J Trop Med Hyg*. 1972;21:123-128.

Baker MS, Strunk HK. Medical aspects of Persian Gulf operations: environmental hazards. *Mil Med*. 1991;156(8):381-385.

Baker MS, Strunk HK. Medical aspects of Gulf operations: serious infectious and communicable diseases of the Persian Gulf and Saudi Arabian Peninsula. *Mil Med*. 1991;156(8):385-390.

Bartelloni PJ, Tesh RB. Clinical and serologic responses of volunteers infected with *phlebotomus* fever virus (Sicilian type). *Am J Trop Med Hyg*. 1976;25:456-462.

Berman JD. Chemotherapy for leishmaniasis: biochemical mechanisms, clinical efficacy, and future strategies. *Rev Infect Dis*. 1988;10:560-586.

Botros BAM, Watts DM, Soliman AK, et al. Serologic evidence of dengue fever among refugees, Hargeyeasa, Somalia. *J Med Virol*. 1989;29:79-81.

Burney MI, Ghafoor A, Saleen M, Webb PA. Casals J. Nosocomial outbreak of viral hemorrhagic fever caused by Crimean hemorrhagic fever-Congo virus in Pakistan, January 1976. *Am J Trop Med Hyg*. 1980;29:941-947.

Carver LA, Flanigan SJ, Connallon PF, Crossley-Miller MK. Epstein-Barr virus infection in Desert Storm reservists. *Mil Med*. 1994;159:580-582.

Centers for Disease Control and Prevention. Dengue fever in U.S. military personnel - Republic of the Phillipines. *MMWR*. 1985;34:495-496, 501-502.

Christie AB. Typhoid and paratyphoid fevers. In: Christie AB, ed. *Infectious Diseases: Epidemiology and Clinical Practice*. New York, NY: Churchill-Livingstone; 1987:100-164.

Clegg JA, Smithers SR, Terry RJ. Acquisition of human antigens by *Schistosoma mansoni* during cultivation *in vitro*. *Nature*. 1971;232:653-654.

Csonka G, Pace J. Endemic nonvenereal treponematosis (Bejel) in Saudi Arabia. *Rev Infect Dis*. 1985;7(Suppl 2):S260-S265.

Other Infectious Disease

Hyams KC, Malone JD, Kapikian AZ, et al. Norwalk virus infection among Desert Storm troops. *J Infect Dis.* 1993;167:986-987.

Hyams KC, Oldfield EC, Scott RM, et al. Evaluation of febrile patients in Port Sudan, Sudan: isolation of dengue virus. *Am J Trop Med Hyg.* 1986;35:860-865.

Iarotski LS, Davis A. The schistosomiasis problem in the world: results of a WHO questionnaire survey. *Bull World Health Organ.* 1981;59:115-127.

Javadian E, Tesh R, Saidi S, Nadim A. Studies on the epidemiology of sandfly fever in Iran. Host-feeding patterns of *Phlebotomus papatasi* in an endemic area of the disease. *Am J Trop Med Hyg.* 1977;26:294-298.

Kambal AM, Mahgoub ES, Jamjoom GA, Chowdhury MN. Brucellosis in Riyadh, Saudi Arabia. A microbiological and clinical study. *Trans R Soc Trop Med Hyg.* 1983;77:820-824.

Kanesa-Thasan N, Iacono-Connors L, Magill A. Dengue serotypes 2 and 3 in US forces in Somalia. *Lancet.* 1994;343:678.

Kapikian AZ, Feinstone SM, Purcell RH, et al. Detection and identification by immune electron microscopy of fastidious agents associated with respiratory illness, acute nonbacterial gastroenteritis, and hepatitis A. *Perspect Virol.* 1975;9:9-47

Karawya EM, Bobo RA. Role of plasmids in the antibiotic resistance of bacteria in Asir Province. *Annals of Saudi Medicine.* 1990;10:165-170.

Kiel FW, Khan MY. Analysis of 506 consecutive positive serologic tests for brucellosis in Saudi Arabia. *J Clin Microbiol.* 1987;25:1384-1387.

Kiel FW, Khan MY. Brucellosis in Saudi Arabia. *Soc Sci Med.* 1989;29:999-1001.

Kuhns DM, Learnard DL. Typhoid and paratyphoid fevers. In: Coates JB, ed. *Preventive Medicine in World War II. Vol. 4. Communicable Diseases.* Washington, DC: Office of the Surgeon General, U.S. Department of the Army; 1958;22:463-476.

Kumar S, al-Hilli F. Bacteremia in Bahrain. A study of aetiological conditions, clinical presentation and antibiotic therapy. *Bahrain Medical Bulletin.* 1988;10:130-140.

Laughlin LW, Legters LJ. Disease threats in Somalia. *Am J Trop Med Hyg.* 1993;48(2):vi-x.

Lazarev VN. Treatment of Crimean hemorrhagic fever patients with convalescent sera. Translation 849 from Department of Medical Zoology, US Naval Medical Research Unit Number Three, Cairo, Egypt. In: Chumakov MP, ed. *Arboviruses. Mater 16 Nauch Sess Inst Polio Virus Entsef.* 1969;2:142-143.

Lehane DE. A seroepidemiologic study of infectious mononucleosis: the development of EB virus antibody in a military population. *JAMA.* 1970;212:2240-2242.

Levine WC, Griffin PM. Vibrio infections on the Gulf coast: results of first year of regional surveillance. Gulf Coast Vibrio Working Group. *J Infect Dis.* 1993;167(2):479-483.

Lindsay GC, Dasey C. Operations Desert Shield/Storm infectious disease rates: a fortuitous anomaly. *United States Army Medical Research and Development Command News.* 1992:February:5-6.

Lo S -C, Buchhols CL, Wear DJ, Holm RC, Marty AM. Histopathology and doxycycline treatment in a previously healthy non-Aids patient systematically infected by *Mycoplasma fermentans* (incongnitus strain). *Modern Pathology.* 1991;6:750-754.

Lubani MM, Lulu AR, Araj GF, Khateeb MI, Qurtom MAF, Dudin KI. Pulmonary brucellosis. *Q J Med.* 1989;71:319-324.

Magzoub M, Kasim AA. Schistosomiasis in Saudi Arabia. *Ann Trop Med Parasitol.* 1980;74:511-513.

Malone JD, Paparello S, Thornton S, Mapes T, Haberberger R, Hyams KC. Parasitic infections in troops returning from Operation Desert Storm. *N Engl J Med.* 1991;325:1448-1449.

Matossian RM, Thaddeus J, Garabedian GA. Outbreak of epidemic typhus in the Northern region of Saudi Arabia. *Am J Trop Med Hyg.* 1963;12:82-90.

McKee, Gasser RA Jr, Magill AJ, Oster CN. *Diagnosis and Treatment of Diseases of Tactical Importance to US CENTCOM Forces 1990.* Falls Church, Va.: Office of the Army Surgeon; 1990.

Meningococcal disease among travelers returning from Saudi Arabia. *MMWR.* 1987;36:559.

Other Infectious Disease

Moaz A, Shannon K, Phillips I. Mechanisms of gentamicin resistance in gram-negative bacilli in Riyadh, Kingdom of Saudi Arabia. *J Antimicrob Chemother*. 1989;24:689-698.

Mohamed AE. The Katayama syndrome in Saudis. *J Trop Med Hyg*. 1985;88:319-322.

Molan AL, Saida LA. Echinococcosis in Iraq: prevalence of *Echinococcus granulosus* in stray dogs in Arbil Province. *Jpn J Med Sci Biol*. 1989;42:137-141.

Moore PS, Reeves MW, Schwartz B, Gellin BG, Broome CV. Intercontinental spread of an epidemic group A *Neisseria meningitidis* strain. *Lancet*. 1989;2:260-263.

Moore PS, Harrison LH, Telzak EE, Ajello GW, Broome CV. Group A meningococcal carriage in travelers returning from Saudi Arabia. *JAMA*. 1988;260:2686-2689.

Mousa AM, Elhag KM, Khogali M, Marafie AA. The nature of human brucellosis in Kuwait: study of 379 cases. *Rev Infect Dis*. 1988;10:211-217.

Nicholson NL, Nicholson GL. The isolation, purification and analysis of specific gene-containing nucleoproteins and nucleoprotein complexes. *Methods Mol Genetics*. 1994;5:281-298.

Nicolson GL, Rosenberg-Nicolson NL. Doxycycline treatment and Desert Storm [letter]. *JAMA*. 1995;237:618-619.

Noah MS, Laajam MA, Hawaas NA, Abdel-Hafez MM. Hydatid liver disease, is it a diagnostic problem? Analysis of 42 consecutive cases in Saudi patients. *Ann Trop Med Parasitol*. 1986;80:607-614.

Novelli VM, Lewis RG, Dawood ST. Epidemic group A meningococcal disease in Haj pilgrims. *Lancet*. 1987;2:863.

Oldfield EC, Rodier GR, Gray GC. The endemic infectious diseases of Somalia. *Clin Infect Dis:*1993; 16 (suppl 3):S132-S157.

Oldfield EC III, Wallace MR, Hyams KC, Yousif AA, Lewis DE, Bourgeois AL. Endemic infectious diseases of the Middle East. *Rev Infect Dis*. 1991;13(suppl 3):S199-S217.

Pak TP, Zykov MF, Mickhailova LI. Contact infections with Crimean hemorrhagic fever in Tadzhik, SSR. *Sov Med*. 1975;1:153-154.

Playfair JH, Blackwell JM, Miller HR. Modern vaccines. Parasitic diseases. *Lancet*. 1990;335(8700):1263-1266.

Qadri SM, Rizvi WH, Rahman SA, al-Dayel F, Flournoy DJ. Saudi Arabian-American differences in antimicrobial resistance of *Escherichia, Klebsiella, and Pseudomonas*. *J Natl Med Assoc*. 1989;81:1061-1064.

Radwan AI, Asmar JA, Frerichs WM, Bekairi SI, Al-Mukayel AA. Incidence of brucellosis in domestic livestock in Saudi Arabia. *Br J Ophthalmol*. 1990;74:249-250.

Ramia S, el-Hazmi MA, Vivian PA, Waller DK, Mushahwar IK, Frosner GG. Delta agent infection in Riyadh, Saudi Arabia. *Trans R Soc Trop Med Hyg*. 1987;81:317-318.

Ramia S, Hossain A, Bakir TM, Waller DK, Vivian PA. Prevalence and subtype of hepatitis B surface antigen (HgsAg) in the Saudi population. *Trop Geogr Med*. 1986;38:63-69.

Ramirez CA, Bran JL, Mejia CR, Garcia JF. Open, prospective study of the clinical efficacy of ciprofloxacin. *Antimicrob Agents Chemother*. 1985;28: 128-132.

Ravel R. Viral and rickettsial infectious diseases. In: *Clinical Laboratory Medicine: Clinical Application of Laboratory Data*. Ed. 5. Chicago, IL: Year Book Medical Publishers; 1989:244-275.

Richards AL, Malone JD, Sheris S, et al. Arbovirus and rickettsial infections among combat troops during Operation Desert Shield/Desert Storm [letter]. *J Infect Dis*. 1993;168:1080-1081.

Ruben B, Band JD, Wong P, Colville J. Person-to-person transmission of *Brucella melitensis*. *Lancet*. 1991;337:14-15.

Sabin AB, Paul JR. *Phlebotomus* (Pappataci or sandfly) fever: a disease of military importance. *JAMA*. 1944;125:603-606.

Saidi S, Tesh R, Javadian E, Sahabi Z, Nadim A. Studies on the epidemiology of sandfly fever in Iran. II. The prevalence of human and animal infection with five *phlebotomus* fever virus serotypes in Isfahan province. *Am J Trop Med Hyg*. 1977;26:288-293.

Saleh AS, Hassan A, Scott RMN, Mellick PW, Oldfield EC, Podgore JK. Dengue in northeast Africa. *Lancet*. 1985;ii:211-212.

OTHER PSYCHIATRIC DISEASE

Adams-Silvan A, Silvan M. `A dream is the fulfillment of a wish': Traumatic dream, repetition compulsion, and the pleasure principle. *Int J Psychoanal.* 1990;71(3):513-522.

Alderman C. When the troops return. *Nurs Stand.* 1991;5(25):19.

Atteberry LR. Neither shall they learn war any more. Health considerations for Desert Storm veterans. *N C Med J.* 1991;52(9):453-457.

Baddoura C. Mental health and war in Lebanon. *Bull Acad Natl Med.* 1990;174(5):583-90.

Barsky AJ, Goodson JD, Lane RS, Cleary PD. The amplification of somatic symptoms. *Psychosom Med.* 1988;50:510-519.

Bartone PT, Ursano RJ, Wright KM, Ingraham LH. The impact of a military air disaster on the health of assistance workers: A prospective study. *J Nerv Ment Dis.* 1989;177:317-328.

Baum A. Stress, intrusive imagery, and chronic distress. *Health Psychol.* 1990;9(6):653-675.

Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review.* 1988;8:77-100.

Bendor A, Gelkopf M, Sigal M. Insanity and war: the Gulf War and a psychiatric institution. *Am J Psychother.* 1993;47(3):424-442.

Ben-Zur H, Zeidner M. Sex differences in anxiety, curiosity and anger: a cross-cultural study. *Sex-Roles.* 1988;19:335-347.

Ben-Zur H, Zeidner M. Anxiety and bodily symptoms under the threat of missile attacks: The Israeli scene. *Anxiety Research.* 1991;4:79-95.

Billings AG, Cronkite RC, Moos RH. Social-environmental factors in unipolar depression: comparison of depressed patients and nondepressed controls. *J Abnorm Psychol.* 1983;92:119-133.

Bleich A, Dycian A, Koslowsky M, Solomon Z, Wiener M. Psychiatric implications of missile attacks on a civilian population. Israeli lessons from the Persian Gulf War. *JAMA.* 1992;268(5):613-615.

Bleich A, Kron S, Margalit C, Inbar G, Kaplan Z, Cooper S, Solomon Z. Israeli psychological casualties of the Persian Gulf war: characteristics, therapy, and selected issues. *Isr J Med Sci.* 1991;27(11-12):673-676.

Blow FC, Cook CA, Booth BM, Falcon SP, Friedman MJ. Age-related psychiatric comorbidities and level of functioning in alcoholic veterans seeking outpatient treatment. *Hosp Community Psychiatry.* 1992;43(10):990-995.

Borus JR. Reentry: I. Adjustment issues facing the returnee. *Arch Gen Psychiatry.* 1973;28:501-506.

Breznitz S. *Cry Wolf: The Psychology of False Alarms.* Hillsdale, Nj: Lawrence Erlbaum; 1984.

Brom D, Witztum E. Recent trauma in psychiatric outpatients. *Am J Orthopsychiatry.* 1992;62(4):545-551.

Burkle FM. Triage of disaster-related neuropsychiatric casualties. *Emerg Med Clin North Am.* 1991;9:87-105.

Burvill PW. The epidemiology of psychological disorders in general medical settings. In: Sartorius N, Goldberg D, de Girolamo G, Costa e Silva J, Lacrubier Y, Wittchen W, eds. *Psychological Disorders in General Medical Settings.* New York, Ny: Hografe & Huber Publishers; 1990:9-20.

Chu JA, Dill DL. Dissociative symptoms in relation to childhood physical and sexual abuse. *Am J Psychiatry.* 1990;147:887-892.

Coggins CC, Zibelin JC, Nelson LF, Hannon T. Inpatient treatment planning. *Suicide Life Threat Behav.* 1993;23(2):162-163.

Cohen J. Things I have learned (so far). *Am Psychol.* 1990;45:1304-1312.

Cohen S, Williamson GM. Stress and infectious disease in humans. *Psychol Bull.* 1991;109:5-24.

Costa PT Jr, McCrae PR. Neuroticism, somatic complaints and disease: Is the bark worse than the bite? *Journal of Personality.* 1987;55:299-316.

Cusack JR. The wounds of war [editorial]. *Am J Psychiatry.* 1993;150(7):997-999.

Decoufle P, Holmgreen P, Boyle CA, Stroup NE. Self-reported health status of Vietnam veterans in relation to perceived exposure to herbicides and combat. *Am J Epidemiol.* 1992;135(3):312-323.

Other Infectious Disease

Scott DA, Burans JP, al-Ouzeib HD, et al. A seroepidemiological survey of viral hepatitis in the Yemen Arab Republic. *Trans R Soc Trop Med Hyg.* 1990;84:288-291.

Sebai ZA. Malaria in Saudi Arabia. *Trop Doct.* 1988;18:183-188.

Sharp TW, Wallace MR, Hayes CG, et al. Dengue fever in U.S. troops during Operation Restore Hope, Somalia, 1992-1993. *Am J Trop Med Hyg.* 1995;53(1):89-94.

Shobokshi OA, Serebour FE. The aetiology of acute viral hepatitis in the western region of Saudi Arabia. *Trans R Soc Trop Med Hyg.* 1987;81:219-221.

Shweiki HM, Hira PR, Behbehani K. Cystic hydatid disease: aspects of the incidence in man in Kuwait, Arabian Gulf. *Eur J Epidemiol.* 1990;6:15-19.

Sigurdsson B, Gudmundsson KR. Clinical findings six years after outbreak of Akureyri Disease. *Lancet.* 1956;1:766-767.

Simmons JS, St. John JH, Reynolds FHK. Experimental studies of dengue. *Philipp J Sci.* 1931;44:1-18.

Strunk HK. *Guidebook of Infectious and Communicable Diseases and Other Health Hazards of the Arabian Peninsula.* Pearl Harbor, HI: U.S. Naval Forces Central Command; 1984.

Suleiman MN, Muscat-Baron JM, Harries JR, et al. Congo/Crimean haemorrhagic fever in Dubai: an outbreak at the Rashid Hospital. *Lancet.* 1980;2:939-941.

Swanepoel R, Gill DE, Shepherd AJ, Leman PA, Mynhardt JH, Harvey S. The clinical pathology of Crimean-Congo hemorrhagic fever. *Rev Infect Dis.* 1989;11(suppl 4):S794-S800.

Tauxe RV, Puhr ND, Wells JG, Hargrett-Bean N, Blake PA. Antimicrobial resistance of *Shigella* isolates in the USA: the importance of international travelers. *J Infect Dis.* 1990;162:1107-1111.

Tesh RB. A method for the isolation and identification of dengue viruses, using mosquito cell cultures. *Am J Trop Med Hyg.* 1979;28:1053-1059.

Tesh RB. The epidemiology of *Phlebotomus* (sandfly) fever. *Isr J Med Sci.* 1989; 25:214-217.

Tesh RB, Chaniotis BN. Transovarial transmission of viruses by phlebotomine sandflies. *Ann N Y Acad Sci.* 1975;266:125-134.

Tesh RB, Saidi S, Gajdamovic SJ, Rodhain F, Vesenjak-Hirjan J. Serological studies on the epidemiology of sandfly fever in the Old World. *Bull World Health Organ.* 1976;54:663-674.

van de Wal BW, Joubert JR, van Eeden PJ, King JB. A nosocomial outbreak of Crimean-Congo haemorrhagic fever at Tygerbert Hospital. Part IV. Preventive and prophylactic measures. *S Afr Med J.* 1985;68:729-732.

Van Eeden PJ, van Eeden SF, Joubert JR, King JB, van de Wal BW, Mitchell WL. A nonsocomail outbreak of Crimean-Congo haemorrhagic fever at Tygerberg Hospital: management of patients. *S Afr Med J.* 1985;68:718-721.

van Eeden PJ, Joubert JR, van de Wal BW, King JB, de Kock A, Groenewald JH. A nosocomial outbreak of Crimean-Congo haemorrhagic fever at Tygerberg Hospital: clinical features. *S Afr Med J.* 1985;68:711-717.

Vassallo L, Khan MA, Edeson JF. Epidemiological aspects and clinical implications of malaria as seen in Jeddah, Saudi Arabia. *Ann Trop Med Parasitol.* 1985;79:349-355.

Vassilenko SM, Vassilev TL, Bozadjiev LG, Bineva IL, Kazarov GZ. Specific intravenous immunoglobulin for Crimean-Congo haemorrhagic fever. *Lancet.* 1990;335:791-792.

Report of a WHO Expert Committee. Viral haemorrhagic fevers. *World Health Organization Tech Rep Ser.* 1985;721:1-126.

Watts DM, Ussery MA, Nash D, Peters CJ. Inhibition of Crimean-Congo hemorrhagic fever viral infectivity yields in vitro by ribavirin. *Am J Trop Med Hyg.* 1989;41:581-585.

West SG. Rheumatic disorders during Operation Desert Storm [letter]. *Arthritis and Rheumatism.* 1993;36(10):1487.

Woolf B. On estimating the evaluation between blood group and disease. *Ann Hum Genet.* 1955;19:251-253.

Other Psychiatric Disease

De Groen JH, op den Velde W, Hovens JE, Falger PR, Schouten EG, Van Duijn H. Snoring and anxiety dreams. *Sleep.* 1993;16(1):35-36.

Derogatis LR, Lipman RS, Rickels K, Uhlenhuth EH, Covi L. The Hopkins Symptom Checklist (HSCL): A measure of primary symptom dimensions. In P. Pichot, ed. *Psychological Measurements in Psychopharmacology: Modern Problems in Pharmacopsychiatry* Basel, Switzerland: Karger; 1974;7:79-110.

Derogatis LR, Spencer PM. The Brief Symptom Inventory (BSI): Administration, scoring & procedures manual. In: *Clinical Psychometric Research.* Baltimore, Md; 1982.

Easton JA, Turner SW. Detention of British citizens as hostages in the Gulf--health, psychological, and family consequences. *BMJ.* 1991;303(6812):1231-1234.

Eberly RE, Engdahl BE. Prevalence of somatic and psychiatric disorders among former prisoners of war. *Hosp Community Psychiatry.* 1991;42(8):807-813.

Eisen SA, Goldberg J, True WR, Henderson WG. A co-twin control study of the effects of the Vietnam war on the self-reported physical health of veterans. *Am J Epidemiol.* 1991;134(1):49-58.

Elder G, Clipp EC. Combat experience and emotional health: impairment and resilience in later life. *J Pers.* 1989;57:311-341.

Engdahl BE, Speed N, Eberly RE, Schwartz J. Comorbidity of psychiatric disorders and personality profiles of American World War II prisoners of war. *J Nerv Ment Dis.* 1991;179(4):181-187.

Farberow NL, Kang HK, Bullman TA. Combat experience and postservice psychosocial status as predictors of suicide in Vietnam veterans. *J Nerv Ment Dis.* 1990;178(1):32-37.

Figley CR. Psychosocial adjustment among Vietnam veterans: an overview of the research. In: CR Figley, ed. *Stress Disorders Among Vietnam Veterans: Theory, Research, and Treatment.* New York, Ny: Brunner/Mazel; 1978:57-70.

Franklin RB, Ebner DG, Xenakis SN, Balson PM. Psychological reactions during chemical warfare training. *Mil Med.* 1983;148:32-235.

Friedman MJ, Charney DS, Southwick SM. Pharmacotherapy for recently evacuated military casualties. *Mil Med.* 1993;158(7):493-497.

Garland FN. Combat stress control in the post-war theater: mental health consultation during the redeployment phase of Operation Desert Storm. *Mil Med.* 1993;158(5):334-338.

Garland FN, Robichaud MR. Knowledge of battle fatigue among division combat medics, and the effectiveness of training. *Mil Med.* 1987;152:608-612.

Gaskell C, Cremin T. The Gulf War. Bringing back memories. *Nurs Times.* 1992;88(14):41-42.

Gerlock AA. Vietnam: returning to the scene of the trauma. *J Psychosoc Nurs Ment Health Serv.* 1991;29(2):4-8.

Green AH. Child maltreatment and its victims: a comparison of physical and sexual abuse. *Psychiatr Clin North Am.* 1988;11:591-610.

Hausman W, Rioch DM. Military psychiatry. *Arch Gen Psychiatry.* 1967;16:727-739.

Hendrix C, Schumm W. Reliability and validity of the Abusive Violence Scale. *Psychol Rep.* 1990;66:1251-1258.

Herman JL, Perry JC, van der Kolk B. Childhood trauma in borderline personality disorder. *Am J Psychiatry.* 1989;146:490-495.

Horwitz AV. Help-seeking processes and mental health services. In: D Mechanic, ed. *Improving Mental Health Services: What the Social Services Can Tell Us.* San Francisco, Ca: Jossey-Bass; 1987.

Hovens JE, op den Velde W, Falger PR, Schouten EG, De Groen JH, Van Duijn H. Anxiety, depression and anger in Dutch Resistance veterans from World War II. *Psychother Psychosom.* 1992;57(4):172-179.

Hudson CJ. The first case of battle hysteria? [letter]. *Br J Psychiatry.* 1990;157:150.

Hughes S. Inside madness. *BMJ.* 1990;301:1476-1478.

Jacob M, Frank E, Kupfer DJ, Carpenter LL. Recurrent depression: an assessment of family burden and family attitudes. *J Clin Psychiatry.* 1987;48:395-400.

Other Psychiatric Disease

Johnson LB, Cline DW, Marcum JM, Intress JL. Effectiveness of a stress recovery unit during the Persian Gulf War. *Hosp Comm Psychiatry.* 1992;43(8):829-831.

Jones FD, Hales RE. Military combat psychiatry: a historical review. *Psychiatric Annals.* 1878;17:525-527.

Jordan BK, Schlenger WE, Hough R, et al. Lifetime and current prevalence of specific psychiatric disorders among Vietnam veterans and controls. *Arch Gen Psychiatry.* 1991;48(3):207-215.

Kaniasty K, Norris FH. Some psychological consequences of the Persian Gulf War on the American people: an empirical study. *Contemporary Social Psychology.* 1991;15:121-126.

Kaplan Z, Kron S, Lichtenberg P, Solomon Z, Bleich A. Military mental health in the Gulf War: the experience at the Central Clinic of the IDF. *Isr J Psychiatry Relat Sci.* 1992;29(1):7-13.

King DW, King LA. Validity issues in research on Vietnam veteran adjustment. *Psychol Bull.* 1991;109(1):107-124.

Klein K, Holt D. The relationship of need for cognition and media credibility to attitudes toward the Persian Gulf War. *Contemporary Social Psychology.* 1991;15:166-171.

Kleinman Y, Korn-Lubetzki I, Eliashiv S, Abramsky O, Eliakim M. High frequency of hemorrhagic strokes in Jerusalem during the Persian Gulf War. *Neurology.* 1992;42(11):2225-2226.

Kroenke K, Spitzer RL, Williams, JBW, et al. Physical symptoms in primary care: predictors of psychiatric disorders and functional impairment. *Arch Fam Med.* 1994;3:774-779.

Lassagne M, Clervoy P, Plouznikoff M, Fajri A. At man's height: a case of war traumatic neurosis. *Med Armees.* 1992;20(3):253-255.

Marlowe DH, Martin JA, Gifford RK. *Observations and Initial Findings of the WRAIR Stress Evaluation Team: Operation Desert Shield: 22 September - 6 October 90.* Washington, DC: Walter Reed Army Institute of Research; 1990.

Miller TW, Martin W, Jay LL. Clinical issues in readaptation for Persian Gulf veterans. *Psychiatr Annals.* 1991;21(11):684-688.

Molgaard CA, Poikolainen K, Elder JP, et al. Depression late after combat: a follow-up of Finnish World War Two veterans from the seven countries east-west cohort. *Mil Med.* 1991;156(5):219-222.

Mollica RF, Wyshak G, Lavelle J, Truong T, Tor S, Yang T. Assessing symptom change in Southeast Asian refugee survivors of mass violence and torture. *Am J Psychiatry.* 1990;147:83-88.

Monajem R. War neurosis according to Chinese medicine: a preliminary survey. *American Journal of Acupuncture.* 1991;19(4):339-344.

Ogata SN, Silk KR, Goodrich S, Lohr NE, Westen D, Hill EM. Childhood sexual and physical abuse in adult patients with borderline disorder. *Am J Psychiatry.* 1990;147:1008-1013.

Ormel J, Koster MWJ, Van Den Brink W, Wan De Villige G. Recognition, management, and course of anxiety and depression in general practice. *Arch Gen Psychiatry.* 1991;48:700-706.

Pendorf JE. Vocational rehabilitation for psychiatric inpatient Vietnam combat veterans. *Mil Med.* 1990;155(8):369-371.

Perconte ST, Dietrick AL, Wilson AT, Spiro KJ, Pontius EB. Psychological and war stress symptoms among deployed and non-deployed reservists following the Persian Gulf war. *Mil Med.* 1993;158:516-521.

Peters J, van Kammen DP, van Kammen WB, Neylan T. Sleep disturbance and computerized axial tomographic scan findings in former prisoners of war. *Compr Psychiatry.* 1990;31(6):535-539.

Pichot T, Rudd D. Preventative mental health in disaster situations: terror on the autobahn. *Mil Med.* 1991;156(10):540-543.

Piekarski AM, Sherwood R, Funari DJ. Personality subgroups in an inpatient Vietnam veteran treatment program. *Psychol Rep.* 1993;72(2):667-674.

Plante TG, Manuel GM. The Persian Gulf War: civilian war-related stress and the influence of age, religious faith, and war attitudes. *J Clin Psychol.* 1992;48:178-182.

Portnoy G, Kantor D, Bar Natan E. Missile attacks and nursing staff: impact of the Gulf War. *Psychosocial Nursing.* 1992;30:21-22.

Other Psychiatric Disease

Pribor EF, Dimwiddie SH. Psychiatric correlates of incest in childhood. *Am J Psychiatry.* 1992;149:52-56.

Rothschild AJ. The dexamethasone suppression test in psychiatric disorders. *Psychiatr Annals.* 1993;23(12):662-670.

Ryan-Wenger NM. Physical and psychosocial impact of activation and deactivation on Army Reserve nurses. *Mil Med.* 1992;157(9):447-452.

Samter J, Fitzgerald ML, Braudaway CA, et al. Debriefing: from military origin to therapeutic application. *J Psychosoc Nurs Ment Health Serv.* 1993;31(2):23-27.

Schnurr PP, Rosenberg SD, Friedman MJ. Change in MMPI scores from college to adulthood as a function of military service. *J Abnorm Psychol.* 1993;102(2):288-296.

Schulberg HC, Burns BJ. Mental disorders in primary care: epidemiologic, diagnostic, and treatment research directions. *General Hospital Psychiatry.* 1988;45:79-87.

Scotti JR, Beach BK, Northrop LME, et al. *An Investigation of the Effects of Involvement in Operation Desert Storm: Psychological Distress in the Veteran, Spouse, and Children.* Presented at the annual convention of the Association for the Advancement of Behavior Therapy. Atlanta, GA; 1993.

Scurfield RM, Tice SN. Interventions with medical and psychiatric evacuees and their families: from Vietnam through the Gulf War. *Mil Med.* 1992;157(2):88-97.

Shipley WC. *Shipley Institute of Living Scale.* Los Angeles, Ca: Western Psychological Services; 1983.

Society for the Psychological Study of Social Issues (SPSSI) Tack Force on Peace. *The Gulf: Historical Perspectives, Psychological Interpretations and Analyses.* Ann Arbor, Mi: SPSSI; 1991;April:1-12

Spielberger CD. *Preliminary manual for the State-Trait Anger Scale (STAS).* Tampa, Fl: University of South Florida Human Resources Institute; 1980.

Spielberger CD, Gorsuch RL, Lushene RE. *STAI: Manual for the State-Trait Anxiety Inventory.* Palo Alto, Ca: Consulting Psychologists Press; 1970.

Stouffer SA, Guttman L, Suchman GA, Lazarsfeld PF, Star SA, Clauser JA. *Studies in Social Psychology in World War II: Measurement and Prediction* (Vol.4). Princeton, Nj: Princeton University Press; 1950.

Stretch RH. Psychosocial readjustment of Canadian Vietnam veterans. *J Consult Clin Psychol.* 1991;59(1):188-189.

Summerfield D, Toser L. 'Low intensity' war and mental trauma in Nicaragua: a study in a rural community. *Med War.* 1991;7(2):84-99.

Sutker PB, Allain AN Jr. MMPI profiles of veterans of WWII and Korea: comparisons of former POWs and combat survivors. *Psychol Rep.* 1991;68(1):279-284.

Sutker PB, Allain AN Jr, Winstead DK. Psychopathology and psychiatric diagnoses of World War II Pacific theater prisoner of war survivors and combat veterans. *Am J Psychiatry.* 1993;150(2):240-245.

Sutker PB, Galina ZH, West JA, Allain AN. Trauma-induced weight loss and cognitive deficits among former prisoners of war. *J Consult Clin Psychol.* 1990;58(3):323-328.

Sutker PB, Winstead DK, Galina ZH, Allain AN. Assessment of long-term psychosocial sequelae among POW survivors of the Korean conflict. *J Pers Assess.* 1990;54(1-2):170-180.

Sutker PB, Winstead DK, Galina ZH, Allain AN. Cognitive deficits and psychopathology among former prisoners of war and combat veterans of the Korean Conflict. *Am J Psychiatry.* 1991;148:67-72.

Sutker PB, Uddo M, Brailey K, Allain AN, Errera P. Psychological symptoms and psychiatric diagnoses in Operation Desert Storm troops serving graves registration duty. *Journal of Traumatic Stress.* 1994;7:159-171.

Sutker PB, Uddo M, Brailey K, Vasterling JJ, Errera P. Psychopathology in war-zone deployed and nondeployed Operation Desert Storm troops assigned graves registration duties. *J Abnorm Psychol.* 1994;103:383-390.

Swanson GS, Blount J, Bruno R. Comprehensive system Rorschach data on Vietnam combat veterans. *J Pers Assess.* 1990;54(1-2):160-169. Published erratum appears in *J Pers Assess.* 1990;55(1-2):391.

Terr LC. Childhood traumas: an outline and overview. *Am J Psychiatry.* 1991;148:10-20.

Thoits PA. Dimensions of life events that influence psychobiological distress: an evaluation and synthesis of the literature. In: H. R. Kaplan (Ed.). *Psychosocial Stress.* New York, Ny: Academic Press; 1983:33-103.

Umberson D, Henderson K. The social construction of death in the Gulf War. *Omega: The Journal of Death and Dying.* 1992;25:1-15.

van der Kolk BA. *Psychological Trauma.* Washington, DC: American Psychiatric Press; 1987.

Vogt T, Pope C, Mullooly J, et al. Mental health status as a predictor of morbidity and mortality: a 15-year follow-up of members of a health maintenance organization. *Am J Public Health.* 1994;84:227-231.

Wolfe J, Brown PJ, Bucsela ML. Symptom responses of female Vietnam veterans to Operation Desert Storm . *Am J Psychiatry.* 1992;149(5):676-679. Published erratum appears in *Am J Psychiatry.* 1992;149(8):1129.

Wolfe J, Brown PJ, Kelley JM. Reassessing war stress: exposure and the Persian Gulf War. *Journal of Social Issues.* 1993;49:15-31.

Wolfe J, Young BL, Brown PJ. *Self-Reported Sexual Assault in Female Gulf War Veterans.* Poster presented at the annual meeting of the Association for Advancement of Behavior Therapy. Boston, Ma; 1992.

OTHER TOXINS AND THEIR TREATMENT

Al-Yakoob SN, Abdal Y, Nasrallah H, al-Majed N. Exposure to particle-bound polyaromatic hydrocarbons in the al-Mansoria residential area during the Kuwait oil fires: a qualitative appraisal of the adsorption role. *Environ Int.* 1993;19(4):319-331.

Al-Yakoob S, Bou-Olayan AH, Bahloul M. Trace metals in gills of fish from the Arabian Gulf. *Bull Environ Contam Toxicol.* 1994;53(5):718-725.

Al-Yakoob SN, Saeed T, al-Hashash H. Polycyclic aromatic hydrocarbons in fish: exposure assessment for Kuwaiti consumers after the Gulf oil spill in 1991. *Environ Int.* 1994;20(2):221-227.

Anderlini VC, Muhammad OS, Zarba MA, Fowler SW, Mirmand P. Trace metals in marine sediments in Kuwait. *Bull Environ Contam Toxicol.* 1982;28:75-80.

Baker EL. Organic solvent neurotoxicity. *Annu Rev Public Health.* 1988;9:223-232. Published erratum appears in *Annu Rev Public Health.* 1989;10: following vii.

Bakir F, Al-Shahristani H, Al-Rawi NY, Khadouri A, Al-Mufti AW. Indirect sources of mercury poisoning in the Iraqi epidemic. *Bull World Health Organ.* 1976;53 (suppl):129-132.

Cole LA. *Clouds of Secrecy, The Army's Germ Warfare Tests Over Populated Areas,* Rowman and Littlefield; 1988:75-104.

Cole LA. Risk and biological defense program. *Physicians for Social Responsibility Quarterly.* 1992;2:40-50.

Davies I. Aluminium, neurotoxicology and dementia. *Rev Environ Health.* 1986;6(1-4):251-296.

Denton GRW, Burdon-Jones C. Trace metals in fish from the Great Barrier Reef. *Mar Pollut Bull.* 1986;17:201-209.

Dorner F, McDonel JL. Bacterial toxin vaccines. *Vaccine.* 1985;3(2):94-102.

Ellis RJ. *Immunobiologic Agents and Drugs Available From the Centers for Disease Control: Descriptions, Recommendations, Adverse Reactions, and Serologic Response.* Atlanta, Ga: Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services; 1982.

Fishbein L. An overview of environmental and toxicological aspects of aromatic hydrocarbons. II. Toluene. *Sci Total Environ.* 1985;42(3):267-288.

Franz DR, Pitt LM, Clayton MA, Hanes MA, Rose KJ. Efficacy of prophylactic and therapeutic administration of antitoxin for inhalation botulism. *Botulinum Tetanus Neurotoxins.* 1993:473-476.

Goodman LJ, Trenholme GM, Kaplan RL, et al. Empiric antimicrobial therapy of domestically acquired acute diarrhea in urban adults. *Arch Intern Med.* 1990;150:541-546.

Gordon JJ, Inns RH, Johnson MK, et al. The delayed neuropathic effects of nerve agents and some other organophosphorus compounds. *Arch Toxicol.* 1983;52(2):71-82.

Griffin MR, Surowiec JJ, McCloskey DI, et al. Foodborne Norwalk virus. *Am J Epidemiol.* 1982;115:178-184.

Other Toxins and their Treatment

Gunderson CH, Lehmann CR, Sidell FR, Jabbari B. Nerve agents: a review. *Neurology*. 1992;42(5):946-950.

Gupta AK. Accumulation of cadmium in the fishes *Heteropneustes fossilis* and *Channa punctatus*. *Environ Ecol*. 1988;6:577-580.

Heggers JP. Microbial invasion—the major ally of war (natural biological warfare). *Mil Med*. 1978;143:390-394.

Heggers VP. Microbial invasion - the major ally of war (natural biological warfare). *Mil Med*. 1978;143:390.

Madany IM, Raveendran E. Polycyclic aromatic hydrocarbons, nickel and vanadium in air particulate matter in Bahrain during the burning of oil fields in Kuwait. *Sci Total Environ*. 1992;116(3):281-289.

Middlebrook JL. Contributions of the U.S. Army to *Botulinum* toxin research. In: Das Gupta BR, ed. *Botulinum and Tetanus Neurotoxins*. New York, NY: Plenum Press; 1993:515-519.

Moen BE, Riise T, Todnem K, Fossan GO. Seamen exposed to organic solvents. A cross-sectional study with special reference to the nervous system. *Acta Neurol Scand*. 1988;78(2):123-135.

Moen BE, Todnem K, Loberg T, Krakenes J, Sleire L, Fossan GO. Cerebellar lesion, polyneuropathy and mental dysfunction in a seaman working on chemical tankers. *Bull Inst Marit Trop Med Gdynia*. 1990;41(1-4):63-67.

NRC (National Research Council). *Biological Markers in Immunotoxicology*. Washington, DC: National Academy Press; 1992.

NRC. *Environmental Neurotoxicology*. Washington, DC: National Academy Press; 1992.

Orma PS, Middleton RK. Aerosolized atropine as an antidote to nerve gas. *Ann Pharmacother*. 1992;26(7-8):937-938.

Porter HO. Aviators intoxicated by inhalation of JP-5 fuel vapors. *Aviat Space Environ Med*. 1990;61(7):654-656.

Radhakrishnaiah K. Accumulation of copper in the organs of freshwater fish, *Labeo rohita* (Hamilton), on exposure to lethal and sublethal concentrations of copper. *J Environ Biol*. 1988;9:319-326.

Rappaport SM. Threshold limit values, permissible exposure limits and feasibility: the bases for exposure limits in the United States. *Am J Ind Med*. 1993;23:683-694.

Ray D, Banerjee SK, Chatterjee M. Bioaccumulation of nickel and vanadium in tissues of the catfish *Clarias batrachus*. *J Inorg Biochem*. 1990;38:169-173.

Reid SD, McDonald DG. Metal binding activity of the gills of rainbow trout (*Oncorhynchus mykiss*). *Can J Fish Aquat Sci*. 1991;48:1061-1068.

Roesijadi G, Klerks PL. Kinetic analysis of cadmium binding to metallothionein and other intracellular ligands in oyster gills. *J Exp Zool*. 1989;251:1-12.

Romeo M, Gnassia-Barelli M. *Donax trunculus* and *Venus verrucosa* as bioindicators of trace metal concentrations in Mauritanian coastal waters. *Mar Biol*. 1988;99:223-227.

Rustam H, Von Burg R, Amin-Zaki L, El Hassani S. Evidence for a neuromuscular disorder in methylmercury poisoning. *Arch Environ Health*. 1975 Apr;30(4):190-195.

Sadiq M, Zaidi TH. Metal concentrations in the sediment of the Arabian Gulf of Saudi Arabia. *Bull Environ Contam Toxicol*. 1985;34:565-571.

Saeed T, al-Yakoob S, al-Hashash H, al-Bahloul M. Preliminary exposure assessment for Kuwaiti consumers to polycyclic aromatic hydrocarbons in seafood. *Environ Int*. 1995;21(3):255-263.

Satchell GH. Respiratory toxicology of fishes. In: Weber LJ, ed. *Aquatic Toxicology of Fishes, Vol. 2*. New York, Ny: Raven Press Ltd; 1984:1.

Spencer PS, Schaumburg HH. Organic solvent neurotoxicity. Facts and research needs. *Scand J Work Environ Health*. 1985;11(suppl):53-60.

Spencer PS, Ludolph AC, Kisby GE. Neurologic diseases associated with use of plant components with toxic potential. *Environ Res*. 1993;62(1):106-113.

Veer MP, Bhat UG, Shanmukhappa H. Copper, chromium and manganese in some fishes of Kali Estuary, Karwar. *Fish Technol Soc Fish Technol Cochin*. 1990;27:112-114.

Wong PTS, Siverberg BA, Chau YK, Hodson PV. Lead and the aquatic biota. In: Narigu, ed. *The Biochemistry of Lead in the Environment*. The Netherlands: Elsevier/North-Holland Biomedical Press; 1978:279.

Zatta P. Interaction between Zn^{2+}, Co^{2+}, Mn^{2+} with hemocyanin from *Carcinus maenas*. *Cah Biol Mar*. 1985;26:241-249.

POSTTRAUMATIC STRESS DISORDER

Alfs DS, McClellan TA. A day hospital program for dual diagnosis patients in a VA medical center. *Hosp Community Psychiatry*. 1992;43(3):241-244.

Alroe C. Post-traumatic stress disorder in Australian World War II veterans attending a psychiatric outpatient clinic [letter]. *Med J Aust*. 1993;159(3):212.

Archibald H, Tuddenham R. Persistent stress reaction after combat: a twenty-year follow-up. *Arch Gen Psychiatry*. 1965;12:475-481.

Arnold AL. Outpatient treatment of posttraumatic stress disorder [letter]. *Mil Med*. 1993;158(6):A4-A5.

Arora RC, Fichtner CG, O'Connor F, Crayton JW. Paroxetine binding in the blood platelets of post-traumatic stress disorder patients. *Life Sci*. 1993;53(11):919-928.

Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry*. 1961;4:561-571.

Belenky GL. Varieties of reaction and adaptation to combat experience. *Bull Menninger Clin*. 1987;51:64-79.

Ben-Tovim DI, Potts NL, Dortmans RJ, Weiss AM, Potter R. Acquired hearing impairment and psychiatric disorder amongst Australian Vietnam veterans [letter]. *Lancet*. 1990;336(8710):324.

Bille DA. Road to recovery. Post-traumatic stress disorder: the hidden victim. *J Psychosoc Nurs Ment Health Serv*. 1993;31(9):19-28.

Bisson JI. Automatism and post-traumatic stress disorder. *Br J Psychiatry*. 1993;163:830-832.

Bjorn A, Eriksson T. Released Bosnian prisoners of war. High risk of posttraumatic symptoms. *Lakartidningen*. 1993;90(24):2305-2308. (Swe).

Blair DT, Hildreth NA. PTSD and the Vietnam veteran: the battle for treatment. *J Psychosoc Nurs Ment Health Serv*. 1991;29(10):15-20.

Blake DD, Cook JD, Keane TM. Post-traumatic stress disorder and coping in veterans who are seeking medical treatment. *J Clin Psychol*. 1992;48(6):695-704.

Blanchard EB. Elevated basal levels of cardiovascular responses in Vietnam veterans with PTSD: a health problem in the making? *Journal of Anxiety Disorders*. 1990;4(3):233-238.

Blanchard EB, Kolb LC, Prins A. Psychophysiological responses in the diagnosis of posttraumatic stress disorder in Vietnam veterans. *J Nerv Ment Dis*. 1991;179(2):97-101.

Blanchard EB, Kolb LC, Prins A, Gates S, McCoy GC. Changes in plasma norepinephrine to combat-related stimuli among Vietnam veterans with posttraumatic stress disorder. *J Nerv Ment Dis*. 1991;179(6):371-373.

Boehnlein JK, Sparr LF. Group therapy with WWII ex-POW's: long-term posttraumatic adjustment in a geriatric population. *Am J Psychother*. 1993;47(2):273-282.

Boudewyns PA, Albrecht JW, Talbert FS, Hyer LA. Comorbidity and treatment outcome of inpatients with chronic combat-related PTSD. *Hosp Community Psychiatry*. 1991;42(8):847-849.

Boudewyns P, Hyer L. Physiological response to combat memories and preliminary treatment outcome in Vietnam veteran PTSD patients treated with direct therapeutic exposure. *Behavior Therapy*. 1990;21(1):63-88.

Boudewyns PA, Woods MG, Hyer L, Albrecht JW. Chronic combat-related PTSD and concurrent substance abuse: implications for treatment of this frequent dual diagnosis. *Journal of Traumatic Stress*. 1991;4(4):549-560.

Bradshaw SL Jr, Ohlde CD, Horne JB. The love of war: Vietnam and the traumatized veteran. *Bull Menninger Clin*. 1991;55(1):96-103.

Brandon S. The psychological aftermath of war: battle shock reactions are normal and victims can be helped. *BMJ*. 1991;302(6772):305-306.

Posttraumatic Stress Disorder

Bremner JD, Scott TM, Delaney RC, et al. Deficits in short-term memory in posttraumatic stress disorder. *Am J Psychiatry.* 1993;150(7):1015-1019.

Bremner JD, Southwick S, Brett E, Fontana A, Rosenheck R, Charney DS. Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *Am J Psychiatry.* 1992;149(3):328-332.

Bremner JD, Southwick SM, Johnson DR, Yehuda R, Charney DS. Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. *Am J Psychiatry.* 1993;150(2):235-239.

Bremner JD, Steinberg M, Southwick SM, Johnson DR, Charney DS. Use of the structured clinical interview for DSM-IV dissociative disorders for systematic assessment of dissociative symptoms in posttraumatic stress disorder. *Am J Psychiatry.* 1993;150(7):1011-1014.

Breslau N, Davis GC. Posttraumatic stress disorder in an urban population of young adults: risk factors for chronicity. *Am J Psychiatry.* 1992;149:671-675.

Breslau N, Davis GC. Posttraumatic stress disorder: the etiologic specificity of wartime stressors. *Am J Psychiatry.* 1987;144:578- 583.

Breslau N, Davis GC, Andreski P, Peterson E. Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Arch Gen Psychiatry.* 1991;48:216-222.

Briggs AC. A case of delayed post-traumatic stress disorder with 'organic memories' accompanying therapy. *Br J Psychiatry.* 1993;163:828-830.

Brill NQ, Beebe GW. A follow-up study of war neuroses (VA Medical Monograph). Washington, DC: U.S. Government Printing Office; 1956.

Brodsky L, Doerman AL, Palmer LS, Slade GF, Munasifi FA. Posttraumatic stress disorder: an eclectic approach. *Int J Psychosom.* 1990;37(1-4):89-95.

Brom D, Kleber RJ, Witztum E. The prevalence of posttraumatic psychopathology in the general and the clinical population. *Isr J Psychiatry Relat Sci.* 1992;28(4):53-63.

Brooks N, McKinley B. Crisis in the Gulf. Posttraumatic stress disorder explained. *Nurs Stand.* 1991;5(19):50-51.

Brophy MH. Cyproheptadine for combat nightmares in post-traumatic stress disorder and dream anxiety disorder. *Mil Med.* 1991;156(2):100-101.

Brown GR, Anderson B. Psychiatric morbidity in adult inpatients with childhood histories of sexual and physical abuse. *Am J Psychiatry.* 1991;148:55-61.

Bullman TA, Kang HK, Thomas TL. Posttraumatic stress disorder among Vietnam veterans on the Agent Orange Registry. A case-control analysis. *Annals of Epidemiology.* 1991;1(6):505-512.

Burges-Watson IP, Wilson GV, Hornsby H. War neurosis and associated physical conditions: an exploratory statistical analysis. *Irish Journal of Psychological Medicine.* 1992;9(1):30-36.

Butler RW, Braff DL, Rausch JL, Jenkins MA, Sprock J, Geyer MA. Physiological evidence of exaggerated startle response in a subgroup of Vietnam veterans with combat-related PTSD. *Am J Psychiatry.* 1990;147(10):1308-1312.

Buydens-Branchey L, Noumair D, Branchey M. Duration and intensity of combat exposure and posttraumatic stress disorder in Vietnam veterans. *J Nerv Ment Dis.* 1990;178(9):582-587.

Camp NM. The Vietnam War and the ethics of combat psychiatry. *Am J Psychiatry.* 1993;150(7):1000-1010.

Carlson EB, Rosser-Hogan R. Trauma experiences, posttraumatic stress, dissociation, and depression in Cambodian refugees. *Am J Psychiatry,* 1991;148: 1548-1551.

Carmen E, Rieker PP, Mills T. Victims of violence and psychiatric illness. *Am J Psychiatry.* 1984;141:378-383.

Carroll EM, Rueger DB, Donahoe CP, Foy DW. *The Marital Adjustment of Help-Seeking Vietnam Veterans With Posttraumatic Stress Disorder.* Paper presented at the annual meeting of the Western Psychological Association. San Francisco, CA; April 1983.

Casella L, Motta RW. Comparison of characteristics of Vietnam veterans with and without posttraumatic stress disorder. *Psychol Rep.* 1990;67(2):595-605.

Chemtob CM, Bauer GB, Neller G, Hamada R, Glisson C, Stevens V. Post-traumatic stress disorder among Special Forces Vietnam veterans. *Mil Med.* 1990;155(1):16-20.

Posttraumatic Stress Disorder

Choy T, de Bosset F. Post-traumatic stress disorder: an overview. *Can J Psychiatry.* 1992;37(8):578-583.

Cordray SM, Polk KR, Britton BM. Premilitary antecedents of post-traumatic stress disorder in an Oregon cohort. *J Clin Psychol.* 1992;48(3):271-280.

Crocq MA, Hein KD, Duval F, Macher JP. Severity of the prisoner of war experience and post-traumatic stress disorder. *European Psychiatry.* 1991;6(1):39-46.

Dagan Y, Lavie P, Bleich A. Elevated awakening thresholds in sleep stage 3-4 in war-related post-traumatic stress disorder. *Biol Psychiatry.* 1991;30(6):618-622.

Danieli Y. Treating survivors and children of survivors of the Nazi Holocaust. In: F.M. Ochberg, ed. *Posttraumatic Therapy and Victims of Violence.* New York, NY: Brunner/Mazel; 1988:278-294.

Davidson J, Kudler H, Smith R, et al. Treatment of posttraumatic stress disorder with amitriptyline and placebo. *Arch Gen Psychiatry.* 1990;47(3):259-266.

Davidson J, Smith R, Kudler H. Validity and reliability of the DSM-III-R criteria for posttraumatic stress disorder: Experience with a structured interview. *J Nerv Ment Dis.* 1989;177:336-341.

Davidson JR, Foa EB. Diagnostic issues in posttraumatic stress disorder: considerations for the DSM-IV. *J Abnorm Psychol.* 1991;100(3):346-355.

Davidson JR, Kudler HS, Saunders WB, et al. Predicting response to amitriptyline in posttraumatic stress disorder. *Am J Psychiatry.* 1993;150(7):1024-1029.

Davidson JR, Kudler HS, Saunders WB, Smith RD. Symptom and comorbidity patterns in World War II and Vietnam veterans with posttraumatic stress disorder. *Compr Psychiatry.* 1990;31(2):162-170.

Davidson L, Fleming I, Baum A. Posttraumatic stress as a function of chronic stress and toxic exposure. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel; 1986;2:57-77.

Davidson LM, Baum A. Chronic stress and posttraumatic stress disorders. *J Consult Clin Psychol.* 1986;54:303-308.

Davidson S. The clinical effects of massive psychic trauma in families of Holocaust survivors. *Journal of Marital and Family Therapy.* 1980;6:11-21.

DeFazio VJ, Pascucci NJ. Return to Ithaca: a perspective on marriage and love in posttraumatic stress disorder. *Journal of Contemporary Psychotherapy.* 1984;14:76-89.

DeAngelis T. For some Viet vets, Gulf war is traumatic. *APA Monitor.* 1991;April:19-20.

Denny N, Robinowitz R, Penk W. Conducting applied research on Vietnam combat-related post-traumatic stress disorder. *J Clin Psychol.* 1987;43(1):56-66.

Department of Veterans Affairs. *Interventions in Traumatic Stress: VAA/DOD Contingency.* Project Code 91VJ. Indianapolis, In: Fort Benjamin Harrison; December 18-21, 1990.

Department of VeteransAffairs. *Impact of Persian Gulf War on Readjustment Counseling Services for Vietnam Veterans and Implementation of Readjustment Counseling for Persian Gulf Veterans.* White Paper; December 30, 1991.

De Silva P. Post-traumatic stress disorder: cross-cultural aspects. *International Review of Psychiatry.* 1993;5(2-3):217-229.

Dillard ML, Bendfeldt F, Jernigan P. Use of thioridazine in post-traumatic stress disorder. *South Med J.* 1993;6(11):1276-1278.

Dille JR. Will world peace mean less PTSD? *Aviat Space Environ Med.* 1990;61(10):966.

Dobson M. Post-traumatic stress disorder in Australian World War II veterans attending a psychiatric outpatient clinic [letter]. *Med J Aust* 1993;159(3):212.

Engel CC Jr, Engel AL, Campbell SJ, McFall ME, Russo J, Katon W. Posttraumatic stress disorder symptoms and precombat sexual and physical abuse in Desert Storm veterans. *J Nerv Ment Dis.* 1993;181(11):683-688.

Eth S, Pynoos R. Developmental perspective on psychic trauma in childhood. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel: 1985;1:36-52.

Fairbank JA, Hansen DJ, Fitterling JM. Patterns of appraisal and coping across different stressor conditions among former prisoners of war with and without posttraumatic stress disorder. *J Consult Clin Psychol.* 1991;59(2):274-281.

Fajri A, Lebigot F. The man who cries: one case of a Maghrebian war traumatic neurosis. *Med Armees.* 1991;19(3):147-150.

Falger PR, op den Velde W, Hovens JE, Schouten EG, De Groen JH, Van Duijn H. Current posttraumatic stress disorder and cardiovascular disease risk factors in Dutch Resistance veterans from World War II. *Psychother Psychosom.* 1992;57(4):164-171.

Fausman W, White P. Diagnostic and psychopharmacological treatment characteristics of 536 inpatients with posttraumatic stress disorder. *J Nerv Mental Dis.* 1989;177:154-159.

Fesler FA. Valproate in combat-related posttraumatic stress disorder. *J Clin Psychiatry.* 1991;52(9):361-364.

Fichtner CG, Arora RC, O'Connor FL, Crayton JW. Platelet paroxetine binding and fluoxetine pharmacotherapy in posttraumatic stress disorder: preliminary observations on a possible predictor of clinical treatment response. *Life Sci.* 1994;54(3):PL39-44.

Figley CR. Toward a field of traumatic stress. *Journal of Traumatic Stress.* 1988;1:3-16.

Foa EB, Steketee G, Rothbaum BO. Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy.* 1989;20:155-176.

Folkman S. Personal control and coping processes: a theoretical analysis. *J Pers Soc Psychol.* 1984;46:839-852.

Fontana A, Rosenheck R, Brett E. War zone traumas and posttraumatic stress disorder symptomatology. *J Nerv Ment Dis.* 1992;180(12):748-755.

Forman SI, Havas S. Massachusetts' post-traumatic stress disorder program: a public health treatment model for Vietnam veterans. *Public Health Rep.* 1990;105(2):172-179.

Foy DW, Carroll EM. Donahoe CP. Etiological factors in the development of PTSD in clinical samples of Vietnam combat veterans. *J Clin Psychol.* 1987;43:1-27.

Foy DW, Resnick HS, Sipprelle RC, Carroll EM. Premilitary, military and postmilitary factors in the development of combat-related stress disorder. *Behavior Therapist.* 1987;10:3-9.

Frank G. On the use of the Rorschach in the study of PSTD. *J Pers Assess.* 1992;59(3):641-643.

Freehill KM. Critical incident stress debriefing in health care. *Crit Care Clin.* 1992;8(3):491-500.

Funari DJ, Piekarski AM, Sherwood RJ. Treatment outcomes of Vietnam veterans with posttraumatic stress disorder. *Psychol Rep.* 1991;68(2):571-578.

Gersons BP, Carlier IV. Post-traumatic stress disorder: the history of a recent concept. *Br J Psychiatry.* 1992;161:742-748.

Gleser GC, Green BL, Winget CN. *Prolonged Psychosocial Effects of Disaster: A Study of Buffalo Creek.* New York, Ny: Academic Press; 1981.

Glover H. Four syndromes of posttraumatic stress disorder: stressors and conflicts of the traumatized with special focus of the Vietnam combat veteran. *Journal of Traumatic Stress.* 1988;1:57-78.

Goldberg J, True WR, Eisen SA, Henderson WG. A twin study of the effects of the Vietnam War on posttraumatic stress disorder. *JAMA.* 1990;263(9):1227-1232.

Goldberg J, True WR, Eisen SA, Henderson WG. Posttraumatic stress reactions in Vietnam veterans. *Dtsch Med Wochenschr* 1990;115(33):1255.

Green BL. Defining trauma: Terminology and generic stressor dimensions. *Journal of Applied Social Psychology.* 1990;20:1632-1642.

Green BL. *Disasters and PTSD* (Review paper for DSM-IV Advisory Commitee on PTSD). Washington, DC: American Psychiatric Association; 1990.

Green BL. Evaluating the effects of disaster. *Psychological Assessment: Journal Consult Clin Psychol.* 1991;3:538-546.

Green BL, Grace MC, Lindy JD, Gleser GC. War stressors and symptom persistence in posttraumatic stress disorder. *Journal of Anxiety Disorders.* 1990;4(1):31-40.

Green BL, Grace MC, Lindy JD, Gleser GC, Leonard A. Risk factors for PTSD and other diagnoses in a general sample of Vietnam veterans. *Am J Psychiatry.* 1990;147(6):729-733.

Posttraumatic Stress Disorder

Green BL, Grace MC, Lindy JD, Leonard AC. Race differences in response to combat stress. *Journal of Traumatic Stress*. 1990;3(3):379-393.

Green BL, Lindy JD, Grace MC. Long-term coping with combat stress. *Journal of Traumatic Stress*. 1988;1:399-412.

Green BL, Lindy JD, Grace MC, Gleser GC. Multiple diagnosis in posttraumatic stress disorder: the role of war stressors. *J Ment Dis*. 1989;177:329-335.

Green BL, Lindy JD, Grace MC, Leonard AC. Chronic posttraumatic stress disorder and diagnostic comorbidity in a disaster sample. *J Nerv Ment Dis*. 1992;180(12):760-766.

Green BL, Wilson JP, Lindy J. Conceptualizing post-traumatic stress disorder: a psychological framework. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder*. New York, Ny: Brunner/Mazel; 1985; Volume 1.

Gruber M, Byrd R. Post-traumatic stress disorder and GI endoscopy: a case study. *Gastroenterol Nurs*. 1993;16(1):17-20.

Gurvits TV, Lasko NB, Schachter SC, Kuhne AA, Orr SP, Pitman RK. Neurological status of Vietnam veterans with chronic posttraumatic stress disorder. *J Neuropsychiatry Clin Neurosci*. 1993;5(2):183-188.

Hamner MB. PTSD and cocaine abuse [letter]. *Hosp Community Psychiatry*. 1993;44(6):591-592.

Hamner MB, Hitri A. Plasma beta-endorphin levels in post-traumatic stress disorder: a preliminary report on response to exercise-induced stress. *J Neuropsychiatry Clin Neurosci*. 1992;4(1):59-63.

Hartman WL, Clark ME, Morgan MK, et al. Rorschach structure of a hospitalized sample of Vietnam veterans with PTSD. *J Pers Assess*. 1990;54(1-2):149-159.

Helzer JE, Robins LN, McEvoy L. Posttraumatic stress disorder in the general population: Findings of the epidemiologic catchment area survey. *N Engl J Med*. 1987;317:1630-1634.

Hendin H, Haas AP. Suicide and guilt as manifestations of PTSD in Vietnam combat veterans. *Am J Psychiatry*. 1991;148(5):586-591.

Hierholzer R, Munson J, Peabody C, Rosenberg J. Clinical presentation of PTSD in World War II combat veterans. *Hosp Community Psychiatry*. 1992;43(8):816-820.

Hobfoll SE. Personal and social resources and the ecology of stress resistance. In: P. Shaver, ed. *Review of Personality and Social Psychology*. Beverly Hills, Ca: Sage; 1985;Vol. 6:265-290.

Hobfoll SE. Traumatic stress: a theory based on rapid loss of resources. *Anxiety Research*. 1991;4:187-197.

Hobfoll SE, London P. The relationship of self-concept and social support to emotional distress among women during war. *Journal of Social and Clinical Psychology*. 1986;12:87-100.

Hobfoll SE, Spielberger CD, Breznitz S, et al. War-related stress: addressing the stress of war and other traumatic events. *Am Psychol*. 1991;46:848-855.

Holahan CJ, Moos RH. Life stressors, personal and social resources, and depression: a 4-year structural model. *J Abnormal Psychol*. 1991;100:31-38.

Hollingshead AB, Redlich FC. *Social Class and Mental Illness*. New York, Ny: Wiley; 1958.

Holsenbeck LS. "Psych-Force 90": the OM (combat stress) team in the Gulf. *Journal of theU.S. Army Medical Department*. 1992;March/April;32-36.

Horowitz MJ, Solomon GF. Delayed stress response syndromes in Vietnam veterans. In: Figley CR, ed. *Stress Disorders Among Vietnam Veterans*. New York, Ny: Brunner/Mazel: 1978:268-280.

Horowitz MJ, Wilner N, Kaltreider N, Alvarez W. Signs and symptoms of posttraumatic stress disorder. *Arch Gen Psychiatry*. 1980;37:85-92.

Hotujac L. Psychiatric disorders in war and other catastrophic circumstances - posttraumatic stress disorder. *Ljec Vjesn*. 1991;113(7-8):265-268.

Hourani LL, Armenian H, Zurayk H, Affifi L. A population-based survey of loss and psychological distress during war. *Social Sci Med*. 1986;23:269-275.

Hovens JE, Falger PR, op den Velde W, Meijer P, De Groen JH, Van Duijn H. A self-rating scale for the assessment of posttraumatic stress disorder in Dutch resistance veterans of World War II. *J Clin Psychol.* 1993;49(2):196-203.

Hovens JE, Falger PR, op den Velde W, Schouten EG, De Groen JH, Van Duijn H. Occurrence of current posttraumatic stress disorder among Dutch World War II resistance veterans according to the SCID. *Journal of Anxiety Disorders.* 1992;6(2):147-157.

Hume F, Summerfield D. War injury and post-traumatic morbidity [letter]. *Lancet.* 1992;340(8828):1164.

Hyer L, Davis H, Boudewyns P, Woods MG. A short form of the Mississippi Scale for Combat-Related PTSD. *J Clin Psychol.* 1-991;47(4):510-518.

Hyer L, Leach P, Boudewyns PA, Davis H. Hidden PTSD in substance abuse inpatients among Vietnam veterans. *J Subst Abuse Treat.* 1991;8(4):213-220.

Hyer L, McCranie EW, Woods MG, Boudewyns PA. Suicidal behavior among chronic Vietnam theatre veterans with PTSD. *J Clin Psychol.* 1990;46(6):713-721.

Hyer L, Walker C, Swanson G, Sperr S, Sperr E, Blount J. Validation of PTSD measures for older combat veterans. *J Clin Psychol.* 1-992;48(5):579-588.

Hyer L, Woods MG, Summers MN, Boudewyns P, Harrison WR. Alexithymia among Vietnam veterans with posttraumatic stress disorder. *J Clin Psychiatry.* 1990;51(6):243-247.

Ichinowatari N. Post-traumatic stress disorder. *J Nat Defense Med Coll.* 1993;18(2):104-111.

Jiranek D. Use of hypnosis in pain management and posttraumatic stress disorder. *Australian Journal of Clinical and Experimental Hypnosis.* 1993;21(1):75-84.

Jones FD. Psychological effects of combat [letter]. *Am J Psychiatry.* 1991;148(8):1099.

Jongedijk RA. Posttraumatic symptoms in the belles-lettres; can fiction be scientific?. *Tijdschr Psychiatr.* 1993;35(5):287-302.

Jordan BK, Marmar CR, Fairbank JA, et al. Problems in families of male Vietnam veterans with posttraumatic stress disorder. *J Consult Clin Psychol.* 1992;60(6):916-926.

Kaplan Z, Singer Y, Lichtenberg P, Solomon Z, Bleich A. Post-traumatic stress disorder in Israel during the Gulf War. *Isr J Psychiatry Relat Sci* 1992;29(1):14-21.

Kardiner A. *The Traumatic Neuroses of War.* Washington, DC: Paul B. Hoeber; 1941.

Keane TM, Caddell JM, Taylor KL. Mississippi Scale for combat-elated posttraumatic stress disorder: three studies in reliability and validity. *J Consult Clin Psychol.* 1988;56:85-90.

Keane TM, Fairbank JA, Caddell JM, Zimering RT, Taylor KL, Mora CA. Clinical evaluation of a measure to assess combat exposure. *Psychol Assess: J Consult Clin Psychol.* 1989;1:53-55.

Keane T, Fairbank J, Caddell J, Zimmering R, Bender M. A behavioral approach to assessing and treating post-traumatic stress disorder in Vietnam veterans. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel; 1985;1:257-294.

Kenderdine SK, Phillips EJ, Scurfield RM. Comparison of the MMPI-PTSD subscale with PTSD and substance abuse patient populations. *J Clin Psychol.* 1992;48(1):136-139.

Kidson MA, Douglas JC, Holwill BJ. Post-traumatic stress disorder in Australian World War II veterans attending a psychiatric outpatient clinic. *Med J Aust.* 1993;158(8):563-566.

Kinzie JD, Boehnlein JK. Psychotherapy of the victims of massive violence: countertransference and ethical issues. *Am J Psychother.* 1993;47(1):90-102.

Kiser LJ, Heston J, Millsap PA, Pruitt DB. Physical and sexual abuse in childhood: relationship with post-traumatic stress disorder. *J Am Acad Child Adolesc Psychiatry.* 1991;30:776-783.

Kobasa SC. Stressful life events, personality, and health: an inquiry into hardiness. *J Pers Soc Psychol.* 1979;37:1-11.

Kolb LC. PTSD psychopathology and the startle response. *Psychiatr Q.* 1991;62(3):233-250.

Koller P, Marmar CR, Kanas N. Psychodynamic group treatment of posttraumatic stress disorder in Vietnam veterans. *Int J Group Psychother.* 1992;42(2):225-246.

Koretzky MB, Peck AH. Validation and cross-validation of the PTSD subscale of the MMPI with civilian trauma victims. *J Clin Psychol.* 1990;46(3):296-300.

Kormos HR. The nature of combat stress. In: Figley CR, ed. *Stress Disorders Among Vietnam Veterans.* New York, Ny: Brunner/Mazel; 1978:3-22.

Kosten TR, Frank JB, Dan E, McDougle CJ, Gille EL Jr. Pharmacotherapy for posttraumatic stress disorder using phenelzine or imipramine. *J Nerv Ment Dis.* 1991;179(6):366-370.

Kosten TR, Wahby V, Giller E Jr, Mason J. The dexamethasone suppression test and thyrotropin-releasing hormone stimulation test in posttraumatic stress disorder. *Biol Psychiatry.* 1990;28(8):657-664.

Kuhne A, Orr S, Baraga E. Psychometric evaluation of post-traumatic stress disorder: the Multidimensional Personality Questionnaire as an adjunct to the MMPI. *J Clin Psychol.* 1993;49(2):218-225.

Kulka RA, Schlenger WE, Fairbank JA, et al. Assessment of posttraumatic stress disorder in the community: prospects and pitfalls from recent studies of Vietnam veterans. *Psychol Assess: J Consult Clin Psychol.* 1991;3:347-560.

Kulka RA, Schlenger WE, Fairbank JA, et al. *Trauma and the Vietnam War Generation: Report of Findings From the National Vietnam Veterans Readjustment Study.* New York, Ny: Brunner/Mazel; 1990:322 (Brunner-Mazel Psychosocial Stress Series, No. 18).

Labbate LA, Snow MP. Posttraumatic stress symptoms among soldiers exposed to combat in the Persian Gulf. *Hosp Community Psychiatry.* 1992;43(8):831-833.

Lambert MT. Pheochromocytoma presenting as exacerbation of posttraumatic stress disorder symptomology. *Int J Psychiatry Med.* 1992;22(3):265-268.

Laufer RS, Brett E, Gallops MS. Dimensions of posttraumatic stress disorder among Vietnam veterans. *J Nerv Ment Dis.* 1985;173:538-545.

Lazarus RS, DeLongis A. Psychological stress and coping in aging. *Am Psychol.* 1983;38:245-254.

Lazarus RS, DeLongis A, Folkman S, Gruen R. Stress and adaptational outcomes: the problem of confounded measures. *Am Psychol.* 1985;40:770-777.

Lazarus RS, Folkman S. *Stress, Appraisal and Coping.* New York, Ny: Springer; 1984.

Lebigot F, Vallet D, Prouvost C, Buferne R. Demand for care in traumatic war neuroses. *Ann Med Psychol (Paris).* 1991;149(2):131-149.

Lee I. Second International Conference on Wartime Medical Services. *Med War.* 1991;7(2):120-128.

Leetz KL, Martino-Saltzman D, Dean RC. Effects of the Persian Gulf War on veterans with combat-related post-traumatic stress disorder. *Mil Med.* 1993;158(1):19-22.

Lerer B, Bleich A, Bennett ER, Ebstein RP, Balkin J. Platelet adenylate cyclase and phospholipase C activity in posttraumatic stress disorder. *Biol Psychiatry.* 1990;27(7):735-740.

Litz BT, Keane TM, Fisher L, Marx B, Monaco V. Physical health complaints in combat-related porttraumatic stress disorder: a preliminary report. *J Traumatic Stress.* 1992;5:131-141.

Litz BT, Penk WE, Walsh S, et al. Similarities and differences between MMPI and MMPI-2 applications to the assessment of posttraumatic stress disorder. *J Pers Assess.* 1991;57(2):238-253.

Long N, Chamberlain K, Vincent C. The health and mental health of New Zealand Vietnam War veterans with posttraumatic stress disorder. *N Z Med J.* 1992;105(944):417-419.

Lundin T, Otto U. Swedish UN soldiers in Cyprus (UNFICYP): their psychological and social situation. *Psychother Psychosom.* 1992;57(4):187-193.

Macleod AD. Posttraumatic stress disorder in World War Two veterans. *N Z Med J.* 1991;104(915):285-288.

Posttraumatic Stress Disorder

Maloney LJ. Posttraumatic stresses of women partners of Vietnam veterans. *Smith College Studies in Social Work.* 1988;58:1220143.

Marble J, Duperet JP, Vauterin C. Traumatism and legal redress: Sisyphus myth. *Med Armees.* 1992;20(3):257-259.

March JS. The nosology of posttraumatic stress disorder. *Journal of Anxiety Disorders.* 1990;4:61-82.

Marcum JM, Cline DW. Combat stress reactions in Iraqi enemy prisoners of war. *Bull Menninger Clin.* 1993;57(4):479-491.

Marlowe DH. *Research on Psycho-Social and Environmental Stresses and Their Consequences in Operations Desert Shield/Storm and Post Return of Troops to Home Bases.* Presented to the Defense Science Board Task Force on Gulf War Health Effects; Arlington, Va: December 22, 1993.

McCann IL, Pearlman LA. Vicarious traumatization: a framework for understanding the psychological effects of working with victims. *Journal of Traumatic Stress.* 1990;3:131-149.

McCarroll JE, Ursano RJ, Fullerton CS. Symptoms of posttraumatic stress disorder following recovery of war dead. *Am J Psychiatry.* 1993;150(12):1875-1877.

McCarroll JE, Ursano RJ, Fullerton CS, Lundy A. Traumatic stress of a wartime mortuary. Anticipation of exposure to mass death. *J Nerv Ment Dis.* 1993;181(9):545-551.

McCarroll JE, Ursano RJ, Fullerton CS, Wright KM. Community consultation following a major air disaster. *J Community Psychol.* 1992;20:271-275.

McCarroll JE, Ursano RJ, Wright KM, Fullerton CS. Handling bodies after violent death: strategies for coping. *Am J Orthopsychiatry.* 1993;63(2):209-214.

McCormack JK, Patterson TW, Ohlde CD, Garfield NJ, Schauer AH. MMPI configural interpretation as applied to posttraumatic stress disorder in Vietnam veterans. *J Pers Assess.* 1990;54(3-4):628-638.

McCranie EW, Hyer LA, Boudewyns PA, Woods MG. Negative parenting behavior, combat exposure, and PTSD symptom severity: test of a person-event interaction model. *J Nerv Ment Dis.* 1992;180:431-438.

McDougle CJ, Southwick SM. Emergence of an alternate personality in combat-related posttraumatic stress disorder. *Hosp Community Psychiatry.* 1990;41(5):554-556.

McDuff DR, Johnson JL. Classification and characteristics of Army stress casualties during Operation Desert Storm. *Hosp Community Psychiatry.* 1992;43(8):812-815.

McFall ME, Mackay PW, Donovan DM. Combat-related PTSD and psychosocial adjustment problems among substance abusing veterans. *J Nerv Ment Dis.* 1991;179(1):33-38.

McFall ME, Mackay PW, Donovan DM. Combat-related posttraumatic stress disorder and severity of substance abuse in Vietnam veterans. *J Stud Alcohol.* 1992;53(4):357-363.

McFall ME, Murburg MM, Ko GN, Veith RC. Autonomic responses to stress in Vietnam combat veterans with posttraumatic stress disorder. *Biol Psychiatry.* 1990;27(10):1165-1175.

McFall ME, Smith DE, Mackay PW, Tarver DJ. Reliability and validity of Mississippi scale for combat-related posttraumatic stress disorder. *Psychol Assess: J Consult Clin Psychol.* 1990;2:114-121.

McFall ME, Smith DE, Roszell DK, Tarver DJ, Malas KL. Convergent validity of measures of PTSD in Vietnam combat veterans. *Am J Psychiatry.* 1990;147:645-648.

McFall ME, Veith RC, Murburg MM. Basal sympathoadrenal function in posttraumatic distress disorder. *Biol Psychiatry.* 1992;31(10):1050-1056.

McFarlane A. The longitudinal course of posttraumatic morbidity: the range of outcomes and their predictors. *J Nerv Ment Dis.* 1988;176:30-39.

McFarlane AC. The aetiology of post-traumatic morbidity: predisposing, precipitating, and perpetuating factors. *Br J Psychiatry.* 1989;154:221-. 228.

McGuire B. Post-traumatic stress disorder: a review. *Irish Journal of Psychology.* 1990;11(1):1-23.

McMillan I. Gulf War. The invisible wounds. *Nurs Times.* 1991;87(8):16-17.

McNally RJ, Kaspi SP, Riemann BC, Zeitlin SB. Selective processing of threat cues in posttraumatic stress disorder. *J Abnorm Psychol.* 1990;99(4):398-402.

Melick ME, Logue JN, Frederic CJ. Stress and disaster. In: Goldberger L, Breznitz S, eds. *Handbook of Stress: Theoretical and Clinical Aspects.* New York, Ny: The Free Press; 1982:613-630.

Mellman TA, Ramsay RE, Fitzgerald SG. Divergence of PTSD and narcolepsy associated with military trauma. *Journal of Anxiety Disorders.* 1991;5(3):267-272.

Mellman TA, Randolph CA, Brawman-Mintzer O, Flores LP, Milanes FJ. Phenomenology and course of psychiatric disorders associated with combat-related posttraumatic stress disorder. *Am J Psychiatry.* 1992;149(11):1568-1574.

Milgram NA. Social support versus self-sufficiency in traumatic and posttraumatic stress reactions. In: Lerer B, Gershon S, eds. *New Directions in Affective Disorders.* New York, Ny: Springer-Verlag; 1989:455-458.

Milgram NA, Hobfoll SE. Generalizations from theory and practice in war-related stress. In: Milgram NA, ed. *Stress and Coping in Time of War.* New York, Ny: Brunner/Mazel; 1986;316-352.

Miller DJ. Simple phobia as a symptom of posttraumatic stress disorder in a former prisoner of war. *Behav Modif.* 1991;15(2):250-260.

Miller DJ, Goreczny AJ, Perconte ST. Comparison of symptom distress between World War II ex-POWs and Vietnam combat veterans with post-traumatic stress disorder. *Journal of Anxiety Disorders.* 1992;6(1):41-46.

Miller TW. Advances in understanding the impact of stressful life events on health. *Hosp Community Psychiatry.* 1988;39:615-622.

Miller TW. Persian Gulf crisis: clinical issues in readaptation. *Colloquium presentation at Kentucky Psychological Association.* Louisville, Ky: Kentucky Psychological Association; April 13, 1991.

Miller TW, Kraus RF, Kamenchenko P, Krasnienski A. Posttraumatic stress disorder in US and Russian veterans. *Hosp Community Psychiatry.* 1993;44(6):585-587.

Milroy R. Observations on stress reactions as seen in Naval Reserve Fleet Hospital training evolutions. *Mil Med.* 1991;156(4):166-168.

Mosnaim AD, Wolf ME, Maturana P, et al. In vitro studies of natural killer cell activity in posttraumatic stress disorder patients. Response to methionine-enkephalin challenge. *Immunopharmacology.* 1993;25(2):107-116.

Mueser KT, Yarnold PR, Foy DW. Statistical analysis for single-case designs. Evaluating outcome of imaginal exposure treatment of chronic PTSD. *Behav Modif.* 1991;15(2):134-155.

Munley PH, Bains DS, Bloem WD. F Scale elevation and PTSD MMPI profiles. *Psychol Rep.* 1993;73(2):363-370.

Muran EM, Motta RW. Cognitive distortions and irrational beliefs in post-traumatic stress, anxiety, and depressive disorders. *J Clin Psychol.* 1993;49(2):166-176.

Murray JB. Posttraumatic stress disorder: a review. *Genet Soc Gen Psychol Monogr.* 1992;118(3):313-338.

Musaph H. A late posttraumatic stress reaction provoked by the Gulf war. *Tijdschr Psychother.* 1991;17(6):356-361.

Musaph H. The Gulf War as the trigger of a later posttraumatic stress reaction. *Tijdschr Psychother.* 1991;17(6):356-361.

Nagy LM, Morgan CA 3d, Southwick SM, Charney DS. Open prospective trial of fluoxetine for posttraumatic stress disorder. *J Clin Psychopharmacol.* 1993;13(2):107-113.

Nezu AM, Carnevale GJ. Interpersonal problem solving and coping reactions of Vietnam Veterans with post-traumatic stress disorder. *J Abnorm Psychol.* 1987;96:155-157.

Niles DP. War trauma and post-traumatic stress disorder. *Am Fam Physician.* 1991;44(5):1663-1669.

Norris FH. Epidemiology of trauma: frequency and impact of different potentially traumatic events on different demographic groups. *J Consult Clin Psychol.* 1992;60:409-418.

North CS, Smith EM, McCool RE, Lightcap PE. Acute postdisaster coping and adjustment. *Journal of Traumatic Stress.* 1989;2:353-360.

Posttraumatic Stress Disorder

O'Brien LS, Hughes SJ. Symptoms of post-traumatic stress disorder in Falklands veterans five years after the conflict. *Br J Psychiatry.* 1991;159:135-141.

O'Connell M. Stress and the professional: some recent naval engagements. *J R Soc Health.* 1991;111(2):64-66.

Orner RJ. Post-traumatic stress disorders and European war veterans. *Br J Clin Psychol.* 1992;31(4):387-403.

Orner RJ, Lynch T, Seed P. Long-term traumatic stress reactions in British Falklands War veterans. *Br J Clin Psychol.* 1993;32(4):457-459.

Orr SP, Claiborn JM, Altman B, et al. Psychometric profile of posttraumatic stress disorder, anxious, and healthy Vietnam veterans: correlations with psychophysiologic responses. *J Consult Clin Psychol.* 1990;58(3):329-335.

Otto U. Traumatic stress. Combat reactions and post-traumatic stress. The war is not over when peace is declared--it is still alive in people's minds. *Lakartidningen.* 1992;89(40):3290-3291.

Paige SR, Reid GM, Allen MG, Newton JE. Psychophysiological correlates of posttraumatic stress disorder in Vietnam veterans. *Biol Psychiatry.* 1990;27(4):419-430.

Parson ER. Ethnicity and traumatic stress: the intersecting point in psychotherapy. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel; 1985;1:313-337.

Perconte ST, Griger ML. Comparison of successful, unsuccessful, and relapsed Vietnam veterans treated for posttraumatic stress disorder. *J Nerv Ment Dis.* 1991;179(9):558-562.

Perconte ST, Goreczny AJ. Failure to detect fabricated posttraumatic stress disorder with the use of the MMPI in a clinical population. *Am J Psychiatry.* 1990;147(8):1057-1060.

Perconte ST, Wilson AT, Pontius EB, et al. Unit-based intervention for Gulf War soldiers surviving a SCUD missile attack: program description and preliminary findings. *Journal of Traumatic Stress.* 1993;6(2):225-238.

Pitman RK. Posttraumatic obsessive-compulsive disorder: a case study. *Compr Psychiatry.* 1993;34(2):102-107.

Pitman RK. Self-mutilation in combat-related PTSD [letter]. *Am J Psychiatry.* 1990;147(1):123-124.

Pitman RK, Altman B, Macklin ML. Prevalence of posttraumatic stress disorder in wounded Vietnam veterans. *Am J Psychiatry.* 1989;146:667-669.

Pitman RK, Orr SP, Forgue DF, Altman B, de Jong JB, Herz LR. Psychophysiologic responses to combat imagery of Vietnam veterans with posttraumatic stress disorder versus other anxiety disorders. *J Abnorm Psychol.* 1990;99(1):49-54.

Pitman RK, Orr SP, Lasko NB. Effects of intranasal vasopressin and oxytocin on physiologic responding during personal combat imagery in Vietnam veterans with posttraumatic stress disorder. *Psychiatry Res.* 1993;48(2):107-117.

Pitman RK, Orr SP, Lowenhagen MJ, Macklin ML, Altman B. Pre-Vietnam contents of posttraumatic stress disorder veterans' service medical and personnel records. *Compr Psychiatry.* 1991;32(5):416-422.

Pitman RK, Orr SP, Shalev AY. Once bitten, twice shy: beyond the conditioning model of PTSD [editorial]. *Biol Psychiatry.* 1993;33(3):145-146.

Pitman RK, van der Kolk BA, Orr SP, Greenberg MS. Naloxone-reversible analgesic response to combat-related stimuli in posttraumatic stress disorder: a pilot study. *Arch Gen Psychiatry.* 1990;47(6):541-544.

Pollock DA. PTSD and risk of suicide [letter]. *Am J Psychiatry.* 1992;149(1):142-143.

Polovina N, Divac L. Posttraumatic stress disorders and psychotherapeutic approach. *Vojnosanit Pregl.* 1992;49(2):115-125.

Pomerantz AS. Delayed onset of PTSD: delayed recognition or latent disorder? [letter]. *Am J Psychiatry.* 1991;148(11):1609.

Quinault W. A study of the incidence of stress and anxiety related health problems among the dependents of RAF personnel during the Gulf War. *Nurs Pract.* 1992;5(2):12-23.

Rabin C, Nardi C. Treating post traumatic stress disorder couples: a psychoeducational program. *Community Ment Health J.* 1991;27(3):209-224.

Rabinowitz S, Margalit C, Mark M, Solomon Z, Bleich A. Debate reawakened: premorbid factors for soldiers with refractory posttraumatic stress disorder. *Psychol Rep.* 1990;67(3 Pt 2):1363-1366.

Rahe RH. Acute versus chronic psychological reactions to combat. *Mil Med.* 1988;153:365-373.

Rahe RH. Recent life change stress and psychological depression. In: Miller TW, ed. *Stressful Life Events.* Madison, Ct: International Universities Press Inc.; 1989.

Rahe RH. Acute versus chronic post-traumatic stress disorder. *Integr Physiol Behav Sci.* 1993;28(1):46-56.

Rahe RH, Arthur J. Life change and illness studies: Past history and future directions. In: Lolas F, Mayer H, eds. *Perspectives on Stress and Stress-Related Topics.* New York, Ny: Springer-Verlag; 1987:108-125.

Rahe RH, Karson S, Howard NS Jr, Rubin RT, Poland RE. Psychological and physiological assessments on American hostages freed from captivity in Iran. *Psychosom Med.* 1990;52(1):1-16.

Ramchandani D. Distinguishing features of delayed-onset posttraumatic stress disorder. *Bull Menninger Clin.* 1990;54(2):247-254.

Ramsay R. Invited review: post-traumatic stress disorder; a new clinical entity? *J Psychosom Res.* 1990;34(4):355-365.

Resnick HS, Kilpatrick DG, Best CL, Kramer TL. Vulnerability-stress factors in development of posttraumatic stress disorder. *J Nerv Ment Dis.* 1992;180:424-430.

Rise in stress reports to Gulf war helpline. *The Independent.* 1992;February 3.

Risse SC, Whitters A, Burke J, Chen S, Scurfield RM, Raskind MA. Severe withdrawal symptoms after discontinuation of alprazolam in eight patients with combat-induced posttraumatic stress disorder. *J Clin Psychiatry.* 1990;51(5):206-209.

Roberts P. The Gulf War. A gulf apart. *Nurs Times.* 1992;88(14):38-41.

Rosenheck R, Leda C, Gallup P. Combat stress, psychosocial adjustment, and service use among homeless Vietnam veterans. *Hosp Community Psychiatry.* 1992;43(2):145-149.

Rosenheck R, VA Persian Gulf Returnees Working Group. *Returning Persian Gulf Troops: First Year Findings.* West Haven, Ct: Northeast Program Evaluation Center of the National Center for Posttraumatic Stress Disorder; 1992.

Ross MC, Wonders J. An exploration of the characteristics of post-traumatic stress disorder in reserve forces deployed during Desert Storm. *Arch Psychiatr Nurs.* 1993;7(5):265-269.

Roszell DK, McFall ME, Malas KL. Frequency of symptoms and concurrent psychiatric disorder in Vietnam veterans with chronic PTSD. *Hosp Community Psychiatry.* 1991;42(3):293-296.

Rubonis AV, Bickman L. Psychological impairment in the wake of disaster: the disaster-psychopathology relationship. *Psychol Bull.* 1991;109:384-399.

Saigh PA. Verbally mediated childhood post-traumatic stress disorder. *Br J Psychiatry.* 1992;161:704-706.

Sandrick K. Expanded delivery system needed for post-traumatic stress. *Hospitals.* 1990;64(11):44-45.

Satel SL, Becker BR, Dan E. Reducing obstacles to affiliation with alcoholics anonymous among veterans with PTSD and alcoholism. *Hosp Community Psychiatry.* 1993;44(11):1061-1065.

Schnurr PP, Friedman MJ, Rosenberg SD. Premilitary MMPI scores as predictors of combat-related PTSD symptoms. *Am J Psychiatry.* 1993;150(3):479-483.

Scurfield RM, Wong LE, Zeerocah EB. An evaluation of the impact of helicopter ride therapy for in-patient Vietnam veterans with war-related PTSD. *Mil Med.* 1992;157(2):67-73.

Shalev A, Bleich A, Ursano RJ. Posttraumatic stress disorder: somatic comorbidity and effort tolerance. *Psychosomatics.* 1990;31(2):197-203.

Shalev AY, Orr SP, Pitman RK. Psychophysiologic assessment of traumatic imagery in Israeli civilian patients with posttraumatic stress disorder. *Am J Psychiatry.* 1993;150(4):620-624.

Shalev AY, Orr SP, Pitman RK. Psychophysiologic response during script-driven imagery as an outcome measure in posttraumatic stress disorder. *J Clin Psychiatry.* 1992;53(9):324-326.

Shehan CL. Spouse support and Vietnam veterans' adjustment to posttraumatic stress disorder. *Family Relations.* 1987;36:55-60.

Posttraumatic Stress Disorder

Sherwood RJ, Funari DJ, Piekarski AM. Adapted character styles of Vietnam veterans with Posttraumatic Stress Disorder. *Psychol Rep.* 1990;66(2):623-631.

Shulman E. Predicting postcombat PTSD by using premilitary MMPI scores [letter]. *Am J Psychiatry.* 1994;151(1):156-157.

Sierles FS, Chen JJ, Messing ML, Besyner JK,Taylor MA. Concurrent psychiatric illness in non-Hispanic outpatients diagnosed as having posttraumatic stress disorder. *J Nerv Ment Dis.* 1986;174:171-173.

Sledge WH, Boydstun JA, Rahe AJ. Self-concept changes related to war captivity. *Arch Gen Psychiatry.* 1980;37:430-443.

Smaldino A. Substance abuse, nightmares, and the combat veteran with PTSD: a focus on the mourning process. In: Smaldino A, editor. *Psychoanalytic approaches to addiction.* New York: Brunner/Mazel; 1991:28-50.

Smith EM, North CS, McCool RE, Shea JM. Acute postdisaster psychiatric disorders: Identification of persons at risk. *Am J Psychiatry.* 1990;147:202-206.

Solkoff N, Gray P, Keill S. Which Vietnam veterans develop posttraumatic stress disorders? *J Clin Psychol.* 1986;42:687-698.

Solomon SD. Mobilizing social support networks in times of disasters. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel; 1985;2:232-263.

Solomon SD, Gerrity ET, Muff AM. Efficacy of treatments for posttraumatic stress disorder. An empirical review. *JAMA.* 1992;268(5):633-638.

Solomon SD, Maser JD. Defining terms and instruments for assessing traumatic stress. *Journal of Applied Social Psychology.* 1990;20:1623-1631.

Solomon Z. Somatic complaints, stress reaction, and posttraumatic stress disorde: a 3-year follow-up study. *Behav Med.* 1988;14:179-186.

Solomon Z. Twenty years after the Yom Kippur War: the belated recognition of war-induced psychic trauma [editorial]. *Isr J Psychiatry Relat Sci.* 1993;30(3):128-129.

Solomon Z, Avitzur M, Mikulincer M. Coping resources and social functioning following combat stress reactions: a longitudinal study. *Journal of Social and Clinical Psychology.* 1989;8:87-96.

Solomon Z, Benbenishty R. The role of proximity, immediacy, and expectancy in frontline treatment of combat stress reaction among Israelis in the Lebanon war. *Am J Psychiatry.* 1986;143:613-617.

Solomon Z, Bleich A, Koslowsky M, Kron S, Lerer B, Waysman M. Post-traumatic stress disorder: issues of co-morbidity. *J Psychiatr Res.* 1991;25(3):89-94.

Solomon Z, Garb R, Bleich A, Grupper D. Reactivation of combat-related posttraumatic stress disorder. *Am J Psychiatry.* 1987;144:51-55.

Solomon Z, Laor N, Weiler D, Muller UF, Hadar O, Waysman M, Koslowsky M, Ben Yakar M, Bleich A. The psychological impact of the Gulf War: a study of acute stress in Israeli evacuees [letter]. *Arch Gen Psychiatry.* 1993;50(4):320-321.

Solomon Z, Levi G, Waysman M, Fried B, Mikulincer M, Florian V, Bleich A. Secondary traumatization among wives of soldiers with combat stress reaction. *Harefuah.* 1993;124(12):750-756, 796. (Heb).

Solomon Z, Margalit C, Waysman M, Bleich A. In the shadow of the Gulf War: psychological distress, social support and coping among Israeli soldiers in a high risk area. *Isr J Med Sci.* 1991;27(11-12):687-695.

Solomon Z, Mikulincer M. Aftermaths of combat stress reactions: a three-year study. *Br J Clin Psychol.* 1992;31(1):21-32.

Solomon Z, Mikulincer M. Psychological sequelae of war. A 2-year follow-up study of Israeli combat stress reaction casualties. *J Nerv Ment Dis.* 1988;176:264-269.

Solomon Z, Mikulincer M, Avitzur E. Coping, locus of control, social support, and combat-related post-traumatic stress disorder. *J Pers Soc Psychol.* 1988;55:279-285.

Solomon Z, Mikulincer M, Benbenishty R. Locus of control and combat-related post-traumatic stress disorder: the intervening role of battle intensity threat appraisal and coping. *Br J Clin Psychol.* 1989;28:131-144.

Solomon Z, Mikulincer M, Hobfoll S. Objective vs. subjective measurement of stress and social support: the case of combat-related reactions. *J Consult Clin Psychol.* 1987;55:577-583.

Posttraumatic Stress Disorder

Solomon Z, Mikulincer M, Waysman M. Delayed and immediate onset posttraumatic stress disorder. II. The role of battle experiences and personal resources. *Soc Psychiatry Psychiatr Epidemiol.* 1991;26(1):8-13.

Solomon Z, Mikulincer M, Waysman M, Marlowe DH. Delayed and immediate onset posttraumatic stress disorder. I. Differential clinical characteristics. *Soc Psychiatry Psychiatr Epidemiol.* 1991;26(1):1-7.

Solomon Z, Oppenheimer B, Elizur Y, Waysman M. Exposure to recurrent combat stress: can successful coping in a second war heal combat-related PTSD from the past? *Journal of Anxiety Disorders.* 1990;4(2):141-146.

Solomon Z, Oppenheimer B, Elizur Y, Waysman M. Trauma deepens trauma: the consequences of recurrent combat stress reaction. *Isr J Psychiatry Relat Sci.* 1990;27(4):233-241.

Solomon Z, Waysman M, Levy G, et al. From front line to home front: a study of secondary traumatization. *Fam Process.* 1992;31(3):289-302.

Solomon Z, Weisenberg M, Schwarzwald J, Mikulincer M. Post-traumatic stress disorder among frontline soldiers with combat stress reaction: the 1982 Israeli experience. *Am J Psychiatry.* 1987;144:448-454.

Solomon Z, Weisenberg M, Schwarzwald J, Mikulincer M. CSR and PTSD as determinants of perceived self-efficacy in battle. *J Soc Clin Psychol.* 1988;6:356-370.

Southwick SM, Morgan A, Nagy LM, et al. Trauma-related symptoms in veterans of Operation Desert Storm: a preliminary report. *Am J Psychiatry.* 1993;150(10):1524-1528.

Southwick SM, Yehuda R, Giller EL Jr. Characterization of depression in war-related posttraumatic stress disorder. *Am J Psychiatry.* 1991;148(2):179-183.

Southwick SM, Yehuda R, Giller EL Jr. Personality disorders in treatment-seeking combat veterans with posttraumatic stress disorder. *Am J Psychiatry.* 1993;150(7):1020-1023.

Spiegel D, Cardena E. New uses of hypnosis in the treatment of posttraumatic stress disorder. *J Clin Psychiatry.* 1990;51(suppl):39-43.

Stretch RH. Incidence and etiology of post-traumatic stress disorder among active army personnel. *J Appl Soc Psychol.* 1986;16:464-481.

Summerfield D, Hume F. War and posttraumatic stress disorder: the question of social context. *J Nerv Ment Dis.* 1993;181(8):522.

Sutker PB. Introduction to the special section on issues and methods in assessment of posttraumatic stress disorder. *Psychol Assess: J Consult Clin Psychol.* 1991;3:517-519.

Sutker PB, Bugg F, Allain AN Jr. Person and situation correlates of post-traumatic stress disorder among POW survivors. *Psychol Rep.* 1990;66(3 Pt 1):912-914.

Sutker PB, Uddo-Crane M, Allain AN. Clinical and research assessment of posttraumatic stress disorder: A conceptual overview. *Psychol Assess: J Consult Clin Psychol.* 1991;3:520-530.

Sutker PB, Uddo M, Brailey K, Allain AN. War-zone trauma and stress-related symptoms in Operation Desert Shield/Storm returnees. *Journal of Social Issues.* 1993;49:33-49.

Talbert FS, Braswell LC, Albrecht JW, Hyer LA, Boudewyns PA. NEO-PI profiles in PTSD as a function of trauma level. *J Clin Psychol.* 1993;49(5):663-669.

Tennant C, Streimer JH, Temperly H. Memories of Vietnam: post-traumatic stress disorders in Australian veterans. *Aust N Z J Psychiatry.* 1990;24(1):29-36.

True WR, Rice J, Eisen SA, Heath AC, Goldberg J, Lyons MJ, Nowak J. A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Arch Gen Psychiatry.* 1993;50(4):257-264.

Uddo M, Vasterling JJ, Brailey K, Sutker PB. Memory and attention in combat-related post-traumatic stress disorder (PTSD). *Journal of Psychopathology and Behavioral Assessment.* 1993;15(1):43-52.

Vaughan K, Tarrier N. The use of image habituation training with post-traumatic stress disorders. *Br J Psychiatry.* 1992;161:658-664.

Vedrinne J, Daligand L. The evaluation of traumatic psychological problems: concerning the decree of January 10, 1992. *J Med Leg Droit Med.* 1993;36(3-4):251-256.

Viola JM, Hicks R, Porter T. Gulf War veterans with PTSD [letter]. *Mil Med.* 1993;158(3):A4.

Vorob'ev AI. The posttraumatic stress syndrome in war veterans with a history of combat mental trauma. *Voen Med Zh.* 1991;(8):71-74.

Wallis GG. Stress in service families. *Proc Roy Soc Med.* 1968;61:976.

Wardak AW. Psychiatric morbidity in the wake of trauma. In: Van Luyn JB, et al., eds. *Emergency Psychiatry Today; 2nd International Congress on Emergency Psychiatry*; 1991; Amsterdam: Elsevier Science Publishers BV;1992:153-156.

Watson CG, Brown K, Kucala T, Juba M, Davenport EC Jr, Anderson D. Two studies of reported pretraumatic stressors' effect on posttraumatic stress disorder severity. *J Clin Psychol.* 1993;49(3):311-318.

Watson CG, Juba M, Anderson PE, Manifold V. What does the Keane et al. PTSD scale for the MMPI measure? *J Clin Psychol.* 1990;46(5):600-606.

Watson CG, Kucala T, Juba M, Manifold V, Anderson PE, Anderson D. A factor analysis of the DSM-III post-traumatic stress disorder criteria. *J Clin Psychol.* 1991;47(2):205-214.

Watson CG, Kucala T, Manifold V, Vassar P. Childhood stress disorder behaviors in Vietnam veterans who do and do not develop posttraumatic stress disorder. *J Nerv Ment Dis.* 1989;177:92-95.

Watson D, Pennebaker JW. Health complaints, stress, and distress: exploring the central role of negative affectivity. *Psychol Rev.* 1989;96:234-254.

Watson IP. Post-traumatic stress disorder in Australian prisoners of the Japanese: a clinical study. *Aust N Z J Psychiatry.* 1993;27(1):20-29.

Watson IP, Muller HK, Jones IH, Bradley AJ. Cell-mediated immunity in combat veterans with post-traumatic stress disorder. *Med J Aust.* 1993;159(8):513-516.

Weathers FW, Huska JA, Keane T. *The PTSD Checklist—Military Version (PCL-M)* Boston, Ma: National Center for PTSD; 1991.

Wilcox J, Briones D, Suess L. Auditory hallucinations, posttraumatic stress disorder, and ethnicity. *Compr Psychiatry.* 1991;32(4):320-323.

Wilson J, Smith W, Johnson S. A comparative analysis of PTSD among various survivor groups. In: Figley CR, ed. *Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder.* New York, Ny: Brunner/Mazel; 1985;1:142-172.

Wolf ME, Mosnaim AD, Puente J, Ignacio R. Plasma methionine-enkephalin in PTSD [letter]. *Biol Psychiatry.* 1991;29(3):305-307.

Wolfe J. Applying principles of critical incident debriefing to the therapeutic management of acute combat stress. Boston, MA: National Center for PTSD; 1990.

Wolfe J, Keane TM. New perspectives in the assessment and diagnosis of combat-related posttraumatic stress disorder. In: J.P. Wilson, Raphael B, eds. *International Handbook of Traumatic Stress Syndromes.* New York, Ny: Plenum Press; 1993:165-178.

Wright KM, Ursano RJ, Bartone PT, Ingraham LH. The shared experience of catastrophe: an expanded classification of the disaster community. *Am J Orthopsychiatry.* 1990;60(1):35-42.

Yaktin US, Labban S. Traumatic war. Stress & schizophrenia. *J Psychosoc Nurs Ment Health Serv.* 1992;30(6):29-33.

Yehuda R, Lowy MT, Southwick SM, Shaffer D, Giller EL Jr. Lymphocyte glucocorticoid receptor number in posttraumatic stress disorder. *Am J Psychiatry.* 1991;148(4):499-504.

Yehuda R, Southwick SM, Giller EL Jr. Exposure to atrocities and severity of chronic posttraumatic stress disorder in Vietnam combat veterans. *Am J Psychiatry.* 1992;149(3):333-336.

Yehuda R, Southwick S, Giller EL, Ma X, Mason JW. Urinary catecholamine excretion and severity of PTSD symptoms in Vietnam combat veterans. *J Nerv Ment Dis.* 1992;180(5):321-325.

Yitzhaki T, Solomon Z, Kotler M. The clinical picture of acute combat stress reaction among Israeli soldiers in the 1982 Lebanon War. *Mil Med.* 1991;156(4):193-197.

Zeidner M, Ben-Zur H. Individual differences in anxiety, coping, and post-traumatic stress in the aftermath of the Persian Gulf War. *Personality and Individual Differences.* 1994;16(3):459-476.

Zeidner M, Hammer A. Coping with missile attack: resources, strategies, and outcomes. *Journal of Personality.* 1992;60:709-746.

Zeitlin SB, McNally RJ. Implicit and explicit memory bias for threat in post-traumatic stress disorder. *Behav Res Ther.* 1991;29(5):451-457.

Zimering RT, Norris VG, Davenport MP. *Anger in Vietnam Veterans With PTSD: Implications for Long Term Cardiovascular Health.* Paper presented at the Fourth Annual Conference of the Society for Traumatic Stress. Dallas, Tx; 1988, October.

PYRIDOSTIGMINE

Adler M, Deshpande SS, Foster RE, Maxwell DM. Effects of subacute pyridostigmine administration on mammalian skeletal muscle function. *J Appl Toxicol.* 1992;12:25-33.

Barbarino A, Corsello SM, Tofani A, et al. Sexual dimorphism of pyridostigmine potentiation of growth hormone (GH) - releasing hormone-induced GH release in humans. *J Clin Endocrinol Metab.* 1991;73:75-78.

Borland RG, Brennan DH, Nicholson AN, Smith PA. Studies on the possible central and peripheral effects in man of cholinesterase inhibitor (pyridostigmine). *Human Toxicology.* 1985;4:293-300.

Bowman PD, Schuschereba ST, Johnson TW, et al. Myopathic changes in diaphragm of rats fed pyridostigmine bromide subchronically. *Fundam Appl Toxicol.* 1989;13:110-117.

Breyer-Pfaf U, Maier U, Brinkmann AM, Schumm F. Pyridostigmine kinetics in healthy subjects and patients with myasthenia gravis. *Clin Pharmacol Ther.* 1985;37:495-501.

Briggs GC, Freeman RK, Yaffe SJ, eds. *Drugs in Pregnancy and Lactation.* Baltimore, Md: Williams & Wilkins; 1990:543-544.

Cook JE, Wenger CB, Kolka MA. Chronic pyridostigmine bromide administration: side effects among soldiers working in a desert environment. *Mil Med.* 1992;157:250-254.

Dettbarn W-D. Anticholinesterase-induced myonecrosis. In: Ballantyne B, Marrs TC, eds. *Clinical and Experimental Toxicology of Organophosphates and Carbamates.* Oxford: Butterworth; 1992:167-179.

Diruhumber P, French MC, Green DM, Leadbeater L, Stratton JA. The protection of primates against soman poisoning by pretreatment with pyridostigmine. *J Pharmacol.* 1979;31:295-299.

Drachman DB. Myasthenia gravis. *N Engl J Med.* 1994;330:1797-1810.

Ellman GL, Courtney DK, Andres V, Featherstone RM. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochemical Pharmacology.* 1961;7:88-95.

Fenichel GM, Kibler WB, Dettbarn W-D. The effect of immobilization and exercise on acetylcholine-mediated myopathies. *Neurology.* 1974;24:1086-1090.

French MC, Wetherell JR, White PDT. The reversal by pyridostigmine of neuromuscular block produced by soman. *Journal of Pharmacy and Pharmacology.* 1979;31:290-294.

Gall P. The use of therapeutic mixtures in the treatment of cholinesterase inhibition. *Fundam Appl Toxicol.* 1981;1:214-216.

Gebbers J-O, Lotscher M, Kobel W, Portmann R, Laissue J-A. Acute toxicity of pyridostigmine in rats: histological findings. *Arch Toxicol.* 1986;58:271-275.

Glikson M, Achiron A, Ram Z, et al. The influence of pyridostigmine administration on neuromuscular functions - studies in healthy human subjects. *Fundam Appl Toxicol.* 1991;16:288-298.

Gordon JJ, Leadbeater L, Maidment MP. The protection of animals against organophosphate poisoning by pretreatment with a carbamate. *Toxicol Appl Pharmacol.* 1978;43:207-216.

Graham C. *Oral Dose Effects of Pyridostigmine on Human Performance.* Fort Detrick, Md: Proceedings of the 4th Annual Chemical Defense Bioscience Review; 1984.

Gupta RC, Patterson GT, Dettbarn W-D. Mechanisms of toxicity and tolerance to diisopropylphosphofluoridate at the neuromuscular junction of the rat. *Toxicol Appl Pharmacol.* 1986;84:541-550.

Harris L, Stitcher D. Protection against diisopropylfluorophosphate intoxication by pyridostigmine and physostigmine in combination with atropine and mecamylamine. *Arch Pharmacol.* 1984;327:64-69.

Pyridostigmine

Harris LW, Talbot BG, Anderson DR, Lennox WJ, Green MD. Oxime induced decarbamylation and atropine/oxime therapy of guinea pigs intoxicated with pyridostigmine. *Life Sci.* 1987;40:577-583.

Harris LW, Talbot BG, Lennox WJ, Anderson DR. The relationship between oxime-induced reactivation of carbamylated acetylcholinesterase and antidotal efficacy against carbamate intoxication. *Toxicol Appl Pharmacol.* 1989;98:128-133.

Hubert M, Lison D. Study of muscular effects of short-term pyridostigmine treatment in resting and exercising rats. *Hum Exp Toxicol.* 1995;14:49-54.

Husdon CS, Foster RE, Kahng MW. Neuromuscular toxicity of pyridostigmine bromide in the diaphragm, extensor digitorum longus, and soleus muscles of the rat. *Fundam Appl Toxicol.* 1985;5(suppl):S260-S269.

Inns RH, Marrs T. Prophylaxis against anticholinesterase poisoning. In: Ballantyne B, Marrs TC, eds. *Clinical and Experimental Toxicology of Organophosphates and Carbamates.* Oxford: Butterworth; 1992:602-610.

Israeli S, Avgar D, Almog S, et al. The effect of repeated doses of 30 mg pyridostigmine bromide on pilot performance in an A-4 flight simulator. *Aviat Space Environ Med.* 1990;61:430-432.

Keeler JR, Hurst CG, Dunn MA. Pyridostigmine used as a nerve agent pretreatment under wartime conditions. *JAMA.* 1991;266:693-695.

Kolka MA, Burgoon PW, Quigley MD, et al. *Red Blood Cell Cholinesterase Activity and Plasma Pyridostigmine Concentration During Single and Multiple Dose Studies.* U.S. Army Research Institute of Environmental Medicine, Technical Report #T3-91; 1991.

Koplovitz I, Harris LW, Anderson DR, Lennox WJ, Stewart JR. Reduction of pyridostigmine pretreatment of the efficacy of atropine and 2-PAM treatment of sarin and VX poisoning in rodents. *Fundam Appl Toxicology.* 1992;18:102-106.

Lennox WJ, Harris LW, Talbot BG, Anderson DR. Relationship between reversible acetylcholinesterase inhibition and efficacy against soman lethality. *Lif Sci.* 1985;37:793-798.

McNall PG, Wolfson B, Tuazon JG, Siker ES. Use of pyridostigmine for the reversal of neuromuscular junction blockade. *Anesth Analg.* 1969;48:1026-1032.

Neish SR, Carter B. More on Desert Storm [letter]. *JAMA.*1991;266(23):3282-3283.

O'Keane V, Dinan TG. Sex steroid priming effects on growth hormone response to pyridostigmine throughout the menstrual cycle. *J Clin Endocrinol Metab.* 1992;75:11-14.

Ram Z, Molcho M, Danon YL, et al. The effect of pyridostigmine on respiratory function in healthy and asthmatic volunteers. *Isr J Med Sci.* 1991;27(11-12):664-668.

Sarno AP. More on Desert Storm [letter]. *JAMA.* 1991;266(23):3282.

Scadding GK, Havard CWH, Lange MJ, Domb I. The long term experience of thymectomy for myasthenia gravis. *J Neurol Neurosurg Psychiatry.* 1985;48:401-406.

Sharabi Y, Danon YL, Berkenstadt H, et al. Survey of symptoms following intake of pyridostigmine during the Persian Gulf war. *Isr J Med Sci.* 1991;27:656-658.

Solana RP, Gennings C, Carter WH, et al. Evaluation of the efficacy of two carbamates, Physostigmine and Pyridostigmine, when used in conjunction for protection against organophosphate exposure. *Fundam Appl Toxicol.* 1990;15:814-819.

Thompson T, Kewitz H, Pluel O. Estimation of cholinesterase activity in undiluted plasma and erythrocyte as a tool for measuring *in vivo* effect of reversible inhibitors. *J Clin Chem Biochem.* 1988;26:469-475.

Vanneste Y, Lison D. Biochemical changes associated with muscle fibre necrosis after experimental organophosphate poisoning. *Hum Exp Toxicol.* 1993;12:365-370.

Wacks I, Oster JR, Perez GO, Kett DH. Spurious hyperchloremia and hyperbiocarbonatemia in a patient receiving pyridostigmine bromide therapy for myasthenia gravis. *Am J Kidney Dis.* 1990;16:76-79.

Wenger CB, Latzka WA. Effects of pyridostigmine bromide on physiological responses to heat, exercise and hypohydration. *Aviat Space Environ Med.* 1992;63:37-45.

Q FEVER

Aitken ID. Clinical aspects and prevention of Q fever in animals. *Eur J Epidemiol.* 1989;5:420-424.

Aitken ID, Bogel K, Cracea E, et al. Q fever in Europe: current aspects of aetiology, epidemiology, human infection, diagnosis and therapy. *Infection.* 1987;15:323-327.

Amano K, Williams JC. Chemical and immunological characterization of lipopolysaccharides from phase I and phase II *Coxiella burnetii. J Bacteriol.* 1984;160:994-1002.

Brooks RG, Licitra CM, Peacock MG. Encephalitis caused by *Coxiella burnetii. Ann Neurol.* 1986;20:91-93.

Cameron DA. Freedman AR, Wansbrough-Jones MH. Q fever encephalitis. *J Infect.* 1990;20:159-162.

Clark WH. Lennette EH, Railsback OC, Romer MS. Q fever in California. VII. Clinical features in one hundred eighty cases. *Arch Intern Med.* 1951;88:155-67.

D'Angelo LJ, Baker EF, Schlosser W. Q fever in.the United States, 1948-1977. *J Infect Dis.* 1979;139:613-615.

Derrick EH. "Q" fever a new fever entity: clinical features. diagnosis, and laboratory investigation. *Med J Aust.* 1937;11:281-299.

Derrick EH. The course of infection with *Coxiella burnetii. Med J Aust.* 1973;1:1051-1057.

Drancourt M, Raoult D, Xeridat B, Milandre L, Nesri M, Dano P. Q fever meningoencephalitis in five patients. *Eur J Epidemiol.* 1991;7:134-18.

Dupuis G, Peter O, Peacock M, Burgdorfer W, Haller E. Immunoglobulin responses in acute Q fever. *J Clin Microbiol.* 1985;22:484-487.

Dwyer DE, Gibbons VL, Brady LM, Cunningham AL. Serological reaction to *Legionella pneumophila* group 4 in a patient with Q fever. *J Infect Dis.* 1988;158:499-500.

Fergusson RJ, Shaw TRD, Kitchin AH, Matthews MB, Inglis JM, PeuthererJF. Subclinical chronic Q fever. *Q J Med.* 1985;57:669-676.

Ferrante MA, Dolan MJ. Q fever meningoencephalitis in a soldier returning from the Persian Gulf war. *Clin Infect Dis.* 1993;16:489-496.

Gallaher WH. Q fever. *JAMA* 1961;177:187-189.

Gomez-Aranda F, Diaz JP. Acebal MR, Cones LL, Rodriguez AN, Moreno JM. Computed tomographic brain scan findings in Q fever encephalitis. *Neuroradiology.* 1984;26:329-332.

Hackstadt T. Antigenic variation in the phase I lipopolysaccharide of *Coxiella burnetii* isolates. *Infect Immun.* 1986;52:337-340.

Kaplan MM, Bertagna P. The geographical distribution of Q fever. *Bull World Health Organ.* 1955;13:829-860.

Kim JG, Durack DT. Rickettsiae and the central nervous system. In: Scheld WM, Whitley RJ, Durack DT, eds. *Infections ot the Central Nervous System.* New York, Ny; Raven Press; 1991:411-424.

Kimbrough RC III Ormsbee RA, Peacock M. et al. Q fever endocarditis in the United States. *Ann Intern Med.* 1979;91:400-402.

Ladurner G. Stunzner D, Lechner H, Sixl W. Q-fieber-meningoencephalitis: ein falbericht. *Nervenarzt.* 1975;46:274-275.

Leedom JM. Q fever: an update. In: Remmington JS, Swartz MN, eds. *Current Clinical Topics in Infectious Diseases: 1.* New York, Ny: McGraw Hill; 1980:304-331.

Lumio J, Penttinen K, Pettersson T. Q fever in Finland: clinical, immunological and epidemiological findings. *Scand J Infect Dis.* 1981;13:17-21.

Luoto L. Casey ML, Pickens EG. Q fever studies in Montana: detection of asymptomatic infection among residents of infected dairy premises. *Am J Epidemiol.* 1965; 81:356-369.

Marmion BP, Ormsbee RA. Kyrkou M, et al. Vaccine prophylaxis of abattoir-associated Q fever: eight years' experience in Australian abattoirs. *Epidemiol Infect.* 1990;104:275-287.

Marrie TJ. Pneumonia and meningoencephalitis due to *Coxiella burnetii. J Infect Dis.* 1985;11:59-61.

Q Fever

Marrie TJ. Q fever: clinical signs, symptoms and pathophysiology. In: Walker DH, ed. *Biology of Rickettsial Diseases. Vol.* 2. Boca Raton, FL: CRC Press; 1988:1-16.

Marrie TJ. *Coxiella burnetii* (Q fever). In: Mandell GL, Douglas RG Jr, Bennett JE, eds. *Principles and Practice of Infectious Diseases. 3rd ed.* New York, Ny: Churchill Livingstone; 1990:1472-1476.

McDade JE. Fishbein DB. Rickettsiaceae: the rickettsiae. In: Lennette EH, Halonen P, Murphy FA, eds. *Laboratory Diagnosis of Infectious Diseases: Principles and Practice. Vol* 2. *Viral, Rickettsial, and Chlamydial Diseases.* New York, Ny: Springer-Verlag. 1988:864-890.

Ormsbee RA, Bell EJ, Lackman DB, Tallent G. The influence of phase on the protective potency of Q fever vaccine. *J Immunol.* 1964;92:404-412.

Ortuno AD, Maeztu C, Munoz JA, Reigadas R, Rodriquez T, Valdes M. Miller Fisher syndrome associated with Q fever. *J Neurol Neurosurg Psychiatry.* 1990;53:615-616.

Peacock MG, Philip RN, Williams JC, Faulkner RS. Serological evaluation of Q fever in humans: enhanced phase I titers of immunoglobulins G and A are diagnostic for Q fever endocarditis. *Infect Immun.* 1983;41:1089-1098.

Pennington JH, Poole PM, Smith AJK, et al. The occurrence of *Coxiella burnetii* in northwestern England and North Wales: a report from five laboratories of the Public Health Laboratory Service. *J Hyg.* 1969;67:125-133.

Peter O, Dupuis G, Peacock MG, Burgdorfer W. Comparison of enzyme-linked immunosorbent assay and complement fixation and indirect fluorescent-antibody tests for detection of *Coxiella burnetii* antibody. *J Clin Microbiol.* 1987;25:1063-1067.

Powell O. "Q" fever: clinical features in 72 cases. *Aust Ann Med* .1960;9:214-223.

Powell OW, Kennedy KP, Mclver M, Silverstone H. Tetracycline in the treatment of "Q" fever. *Aust Ann Med.* 1962;11:184-188.

Reilly S, Northwood JL, Caul EO. Q fever in Plymouth, 1972-88: a review with particular reference to neurological manifestations. *Epidemiol Infect.* 1990;105:391-408.

Robbins FC, Ragan CA. Q fever in the mediterranean area: report of its occurrence in allied troops. I. Clinical features of the disease. *Am J Hyg.* 1946;44:6-22.

Robbins FC, Rustigian R. Q fever in the mediterranean area: report of its occurrence in allied troops. IV. A laboratory outbreak. *Am J Hyg.* 1946;44:64-71.

Sawyer LA, Fishbein DB, McDade JE. Q fever: current concepts. *Rev Infect Dis.* 1987;9:935-946.

Schuil J, Richardus JH, Baarsma GS, Schaap GJP. Q fever as a possible cause of bilateral optic neuritis. *Br J Ophthalmol.* 1985;69:580-583.

Schwartz RB. Manic psychosis in connection with Q fever. *Br J Psychiatry.* 1974;124:140-143.

Shaked Y, Samra Y. Q fever meningoencephalitis associated with bilateral abducens nerve paralysis, bilateral optic neuritis and abnormal cerebrospinal fluid findings. *Infection.* 1989;17:394-395.

Sobradillo V, Ansola P, Baranda F. Corral C. Q fever pneumonia: a review of 164 community-acquired cases in the Basque country. *Eur Respir J.* 1989;2:263-266.

Spelman DW. Q fever: a study of 111 cases. *Med J Aust.* 1982;1:547-553.

Turck WPG, Howitt G, Turnberg LA, et al. Chronic Q fever. *Q J Med.* 1976;45:193-217.

Van Scoy RE. Culture-negative endocarditis. *Mayo Clin Proc.* 1982;57:149-154.

Wallace SJ, Zealley H. Neurological, electroencephalographic, and virological findings in febrile children. *Arch Dis Child.* 1970;45:611-623.

Wilson HG, Neilson GH, Galea EG, Stafford G, O'Brien MF. Q fever endocarditis in Queensland. *Circulation.* 1976;53:680-684.

Whittick IW. Necropsy findings in a case of Q fever in Britain. *BMJ.* 1950;1:979-980.

Worswick D, Marmion BP. Antibody responses in acute and chronic Q fever and in subjects vaccinated against Q fever. *J Med Microbiol.* 1985;19:281 -296.

REPRODUCTIVE DISEASE

Alberman E, Polani PE, Roberts JAF, et al. Parental exposure to x-irradiation and Down's syndrome. *Ann Hum Genet.* 1972;36:195-206.

Amin-Zaki L, Majeed MA, Elhassani SB, et al. Prenatal methylmercury poisoning: chemical observations over five years. *Am J Dis Child.* 1979;133:172-177.

Annau Z. Organometals and brain development. *Prog Brain Res.* 1988;73:295-303.

Aschengrau A, Monson RR. Paternal military service in Vietnam and the risk of late adverse pregnancy outcomes. *Am J Public Health.* 1990;80(10):1218-1224.

Ascherio A, Chase R, Cote T, et al. Effect of the Gulf War on infant and child mortality in Iraq. *N Engl J Med.* 1992;327:931-936.

Bell JU, Thomas JA. Effects of lead in mammalian reproduction. In: Singhal RL, Thomas JA, eds. *Lead Toxicity.* Baltimore, Md: Urban and Schwarzenberg, Inc.; 1980:169-186.

Berezuk GP, McCarty GE. Investigational drugs and vaccines fielded in support of Operation Desert Storm. *Mil Med.* 1992;157:404-406.

Biddle PG. The role of genetic studies in developmental toxicology. In: Kimmel CA, Buelke-Sam J, eds. *Developmental Toxicology.* New York, Ny: Raven Press; 1981.

Boling RO, Schipul AH Jr, Barnhill DR, Chaney S, Beam TL. Prevalence of neural tube defects in United States Army treatment facilities, 1975-1985; cost analysis of routine screening. *Mil Med.* 1988;153(6):293-295.

Bracken MB. Methodologic issues in the epidemiologic investigation of drug-induced congenital malformations. In: Bracken MB, ed. *Perinatal Epidemiology.* Oxford, England: Oxford University Press; 1984:423-445.

Brandt EN Jr. The CDC study of Vietnam veterans' risks of fathering infants with birth defects [editorial]. *Public Health Rep.* 1984;99(6):529-530.

Brent RL. Radiations and other physical agents. In: Wilson JG, Fraser FC, eds. *Handbook of Teratology. Vol. 1.* New York, Ny: Plenum Press; 1977.

Buffler PA, Aase JM. Genetic risks and environmental surveillance. *J Occup Med.* 1982;24:305-314.

Calle EE, Khoury MJ. Completeness of the discharge diagnoses as a measure of birth defects recorded in the hospital birth record. *Am J Epidemiol.* 1991;134(1):69-77.

Can N. Effects on offspring of paternal exposure to herbicides. In: Westing AH, ed. *Herbicides In War: The Long-Term Ecological and Human Consequences; International Symposium on Herbicides and Defoliants in War: The Long-Term Effects on Man and Nature*; 1983 Jan 13-20; Ho Chi Minh City, Vietnam. Philadelphia, Pa: Taylor and Francis; 1984:137-140.

Cohen BH, Lilienfeld AM, Kramer S, et al. Parental factors in Down's syndrome: results of the second Baltimore case-control study. In: Hook EB, Porter IH, eds. *Population Cytogenetics.* New York, Ny: Academic Press; 1977.

Cohen FL. Paternal contributions to birth defects. *Nurs Clin North Am.* 1986;21(1):49-64.

Colie CF. Male-mediated teratogenesis. *Reprod Toxicol.* 1993;7(1):3-9.

Constable JD, Hatch MC. Reproductive effects of herbicide exposure in Vietnam: recent studies by the Vietnamese and others. *Teratogenesis Carcinog Mutagen.* 1985;5(4):231-250.

Cornblath M, Hartmann AF. Methemoglobinemia in young infants. *J Pediatrics.* 1984;33:421-425.

Cowley G, Hager M, Liu M. Tracking the second storm. *Newsweek.* 1994;May 16:56-57.

Cutler JJ, Parker GS, Rosen S, Prenney B, Healey R, Caldwell GG. Childhood leukemia in Woburn, Massachusetts. *Public Health Rep.* 1986;101:201-205.

Dalterio S, Badr F, Bartke A, et al. Cannabinoids in male mice: effects on fertility and spermatogenesis. *Science.* 1982;216:315-316.

Dan BB. Vietnam and birth defects [editorial]. *JAMA.* 1984;252(7):936-937.

DeStefano F, Amest JL, Kresnos MJ, Schrader SM, Katz DF. Semen characteristics of Vietnam veterans. *Reprod Toxicol.* 1989;3(3):165-174.

Reproductive Disease

Donovan JW, MacLennan R, Adena M. Vietnam service and the risk of congenital anomalies. A case-control study. *Med J Aust.* 1984;140(7):394-397.

Elwood JM, Coldman AJ. Water composition in the etiology of anencephalus. *Am J Epidemiol.* 1981;133:681-690.

Elwood JM, Elwood JH. *Epidemiology of anencephalus and spina bifida.* New York, Ny: Oxford University Press; 1980.

Elwood JM, Elwood JH. International variation in the prevalence at birth of anencephalus in relation to maternal factors. *Int J Epidemiol.* 1982;11:132-137.

Erickson JD, Mulinare J. Vietnam veterans' risk for fathering children with birth defects-reply. *JAMA.* 1985;254:609-610.

Erickson JD, Mulinare J, James LM, Fitch TG. Design and execution of a very large birth-defects case-control study. *Prog Clin Biol Res.* 1985;163B:273-277.

Erickson JD, Mulinare J, McClain PW, et al. Vietnam veterans' risks for fathering babies with birth defects. *JAMA.* 1984;252(7):903-912.

Evans HJ, Fletcher J, Torrance M, et al. Sperm abnormalities and cigarette smoking. *Lancet.* 1981;1:627-629.

Field B, Kerr C. Reproductive behaviour and consistent patterns of abnormality in offspring of Vietnam veterans. *J Med Genet.* 1-988;25(12):819-826.

Gaffey WR. Agent Orange and birth defects. *N Engl J Med.* 1983;309:492.

GAO (U.S. General Accounting Office). *Operation Desert Storm: Questions on Possible Exposure to Reproductive Toxicants.* Washington, DC: GAO; 1994.

GAO Final Report. *Operation Desert Storm: Questions Remans on Possible Exposure to Reproductive Toxins.* GAO/PEMD-94-30. Department of Defense Comments on the GAO Findings, Recommendations, and Rebuttals.

Goldberg J, Eisen SA, True WR, Henderson WG. Health effects of military service. Lessons learned from the Vietnam experience. *Ann Epidemiol.* 1992;2(6):841-853.

Gordon JE, Shy CM. Agricultural chemical use and congenital cleft lip and/or palate. *Arch Environ Health.* 1981;36:213-221.

Greenland S, Staisch KJ, Brown N, et al. The effects of marijuana use on pregnancy. 1. A preliminary epidemiologic study. *Am J Obstet Gynecol.* 1982;143:408-413.

Halberg F. *Biological Rhythms and Endocrine Function: Advances in Experimental Medicine and Biology.* Hedlund L, Franz J, Kenny AD, eds. New York, Ny: Plenum Press; 1973:1-42.

Harris RE. Viral teratogenesis: a review with experimental and clinical perspectives. *Am J Obstet Gynecol.* 1974;119:966-1008.

Health status of Vietnam veterans. III. Reproductive outcomes and child health. The Centers for Disease Control Vietnam Experience Study. *JAMA.* 1988;259(18):2715-2719.

Hegesh E, Shiloah J. Blood nitrates and infantile methemoglobinemia. *Clin Chim Acta.* 1982;125:107-115.

Hemminki K, Lindbohm ML, Taskinen H. Transplacental toxicity of environmental chemicals. Environmental causes and correlates of spontaneous abortions, malformations, and childhood cancer. *Adv Perinatal Med.* 1986;5:43-91.

Hemminki K, Mutanen P, Saloniemi I, et al. Congenital malformations and maternal occupation in Finland: multivariate analysis. *J Epidemiol Community Health.* 1981;35:5-10.

Holmberg PC, Murminen M. Congenital defects of the central nervous system and occupational factors during pregnancy: a case-referent study. *Am J Indust Med.* 1980;1:167-176.

Holmes LB, Driscoll SG, Atkins LA. Etiologic heterogeneity of neural tube defects. *N Engl J Med.* 1976;294:365-369.

Hook EB. Down's syndrome: its frequency in human populations and some factors pertinent to variation in rates. In: de la Cruz FF, Gerald PS, eds. *Trisomy 12 (Down's Symdrome: Research Perspective).* Baltimore, Md: University Park Press; 1981:3-67.

Hook EB. Incidence and prevalence as measures of the frequency of birth defects. *Am J Epidemiol.* 1982;116:743-747.

Hurley LS. Nutritional deficiencies and excesses. In: Wilson JG, Fraser FC, eds. *Handbook of Teratology. Vol 1.* New York, Ny: Plenum Press; 1977.

Janerich DT. Anencephaly and maternal age. *Am J Epidemiol.* 1972;96:319-326.

Janerich DT. Maternal age and spina bifida: longitudinal versus cross-sectional analysis. *Am J Epidemiol.* 1972;96:389-395.

Janerich DT. Epidemic waves in the prevalence of anencephaly and spina bifida in New York State. *Teratology.* 1973;8:253-256.

Janerich DT. Endocrine dysfunction and anencephaly and spina bifida: an epidemiologic hypothesis. *Am J Epidemiol.* 1974;99:1-6.

Janerich DT. Environmental hazards and birth defects. *Ann NY Acad Sci.* 1979;329:10-13.

Janerich DT, Piper JM, Glebatis DM. Oral contraceptives and congenital limb reduction defects. *N Engl J Med.* 1974;291:697-700.

Janerich DT, Piper JM, Glebatis DM. Oral contraceptives and birth defects. *Am J Epidemiol.* 1980;112:73-79.

Janerich DT, Polednak AP. Epidemiology of birth defects. *Epidemiologic Reviews.* 1983;5:16-37.

Johnson RT. Effects of viral infection on the developing nervous system. *N Engl J Med.* 1972;287:599-604.

Kalter H. *Teratology of the Central Nervous System.* Chicago, Il: University of Chicago Press; 1968.

Kaplan KM, Cochi SL, Edmonds LD, et al. A profile of mothers giving birth to infants with congenital rubella syndrome: an assessment of risk factors. *Am J Dis Child.* 1990;144:118-123.

Khoury MJ, Erickson JD, James LM. Etiologic heterogeneity of neural tube defects: clues from epidemiology. *Am J Epidemiol.* 1982;115:538-548.

Kline J, Stein Z, Strobino B, et al. Surveillance of spontaneous abortions: power in environmental monitoring. *Am J Epidemiol.* 1977;106:345-350.

Knightly P, Potter E, Wallace M. *Suffer the Children: The Story of Thalidomide.* New York, Ny: Viking Press; 1979.

Kristensen P, Irgens LM, Daltveit AK, Anderson A. Perinatal outcome among children of men exposed to lead and organic solvents in the printing industry. *Am J Epidemiol.* 1993;137:134-144.

Kurrent JE, Sever JL. Infectious diseases. In: Wilson JG, Fraser FC, eds. *Handbook of Teratology. Vol. 1.* New York, Ny: Plenum Press; 1977.

Lammer EJ, Chen DT, Hoar RM, et al. Retinoic acid embryopathy. *N Engl J Med.* 1985;313:837-841.

LaVecchio FA, Pashayan HM, Singer W. Agent Orange and birth defects [letter]. *N Engl J Med.* 1983;308(12):719-720.

Leck I. Causation of neural tube defects: clues from epidemiology. *Br Med Bull.* 1974;30:158-163.

Long JD, Thomas RL. A one-year experience in the antenatal evaluation of suspected abdominal wall and bowel defects at a military tertiary referral center. *Mil Med.* 1993;158(3):199-202.

Lynberg MC, Khoury MJ, Lu X, Cocian T. Maternal flu, fever, and the risk of neural tube defects: a population-based case-control study. *Am J. Epidemiol.* 1994;140:244-255.

Marshall WC. Clinical aspects of pre and perinatal infection. In: Lambert HP, Wood CBS, eds. *Immunological Aspects of Infection in the Fetus and New-Born.* New York, Ny: Academic Press; 1981.

Mattison DR. *A Framework for Diagnosis: Reproductive and Developmental Toxicity.* Presentation to NIH Technology Assessment Workshop Panel on the Persian Gulf Experience and Health, Bethesda, Md; 1994.

Mississippi State Department of Health. *Mississippi State Department of Health Preliminary Findings on Children of Gulf War Vets. Health Communications and Public Relations.* Jackson, MS; 1994.

Myrianthopoulos NC, Chung CS. Congenital malformations in singletons: epidemiologic survey. In: *Report From the Collaborative Perinatal Project.* New York, NY: Stratton Intercontinental Medical Report Book Corp; 1974.

Nguyen TN, Tran TT, Pham KP. An estimate of reproductive abnormalities in women inhabiting herbicide sprayed and non-herbicide sprayed areas in the south of Vietnam, 1952-1981. *Chemosphere.* 1989;18(1-6):843-846.

Reproductive Disease

Oakley GP Jr. Population and case-control surveillance in the search for environmental causes of birth defects. *Public Health Rep.* 1984;99(5):465-468.

Oakley GP, Flynt JW. Increased prevalence of Down's syndrome (monogolism) among the offspring of women treated with ovulation-inducing agents. *Am J Hum Genet.* 1972;24:20a.

Olshan AF, Kay T, Baird PA. Birth defects among offspring of firemen. *Am J Epidemiol.* 1990;131:312-320.

Olshan AF, Schnitzer PG, Baird PA. Paternal age and risk of congenital heart defects. *Teratology.* 1994;50:80-84.

Olshan AF, Teschke K, Baird PA. Paternal occupation and congenital anomalies in offspring. *Am J Ind Med.* 1991;20:447-475.

Penman AD, Tarver RS, Currier MM. *Suspected Increase of Birth Defects and Health Problems Among Children Born to Persian Gulf War Veterans From Two Mississippi National Guard Units.* Mississippi State Department of Health; 1994.

Picone TA, Allen LH, Olsen PN, et al. Pregnancy outcome in North American women. II. Effects of diet, cigarette smoking, stress and weight gain on placentas, and on neonatal physical and behavioral characteristics. *Am J Clin Nutr.* 1982;36:1214-1224.

Plotz EJ. Virus disease in pregnancy. *NY State J Med.* 1965;65:1239-1251.

Polednak AP, Janerich DT. Uses of available record systems in epidemiologic studies of reproductive toxicology. *Am J Indust Med.* 1983;4:329-348.

Reyna TM. Observations of a pediatric surgeon in the Persian Gulf War. *J Pediatr Surg.* 1993;28(2):209-213.

Rosa C. Spontaneous abortion rate and the Gulf war mobilization. *J US Army Med Dep.* 1993;September-October:P8-8-93-9/10.

Rush D, Stein Z, Susser M. *Diet in Pregnancy: A Randomized Controlled Trial of Nutritional Supplements.* New York, Ny: Alan R. Liss, Inc.; 1980.

Savitz DA, Schwingl PJ, Keels MA. Influence of paternal age, smoking, and alcohol consumption on congenital anomalies. *Teratology.* 1991;44:429-440.

Saxen L, Rapola J. *Congenital Defects.* New York, Ny: Holt, Rinehart & Winston, Inc.; 1969.

Schulman JD, Simpson JL, eds. *Genetic Diseases in Pregnancy: Maternal Effects and Fetal Outcome.* New York, Ny: Academic Press; 1981.

Shepard TH. *Catalog of Teratogenic Agents.* Baltimore, Md: The Johns Hopkins University Press; 1980.

Smith DW. *Recognizable Patterns of Human Deformation.* Philadelphia, Pa: WB Saunders Co.; 1981.

Soyka LF, Joffe JM. Male mediated drug effects on offspring. In: Schwarz RH, Yaffe SJ, eds. *Drug and Chemical Risks to the Fetus and Newborn.* New York, Ny: Alan R. Liss, Inc.; 1980.

Staples RE. Predictiveness and limitations of test methods in teratology. *Environ Health Perspect.* 1976;18:95-96.

Stierman L. Birth defects in California: 1983-1990. In: *California Birth Defects Monitoring Program.* December 1994.

Streissguth AP, Landesman-Dwyer S, Martin JC, et al. Teratogenic effects of alcohol in humans and laboratory animals. *Science.* 1980;209:353-361.

Tippit S. What's wrong with our children? *Ladies' Home Journal.* 1994;June:141-143,148.

Tsubaki T, Irukayama K, eds. *Methylmercury poisoning in Minamata and Niigata, Japan.* Amsterdam: Elsevier Scientific Publishing Co.; 1977.

Vietnam veterans' risks for fathering babies with birth defects. *MMWR .* 1984;33(32):457-459.

Warkany J. History of teratology. In: Wilson JG, Fraser FC, eds. *Handbook of Teratology, Vol. 1.* New York, Ny: Plenum Press; 1977.

Warkany J. Prevention of congenital malformations. *Teratology.* 1981;23:175-189.

Whorton MD. Adverse reproductive outcomes: the occupational health issue of the 1980's. *Am J Public Health.* 1983;73:15-16.

Wilcox AJ, Wienberg CR, O'Connor JE, et al. Incidence of early loss of pregnancy. *N Engl J Med.* 1988;189-194.

Wilson JG. Environmental chemicals. In: Wilson JG, Fraser FC, eds. *Handbook of Teratology. Vol. 1.* New York, Ny: Plenum Press; 1977.

Wolfe WH, Michalek JE, Miner JC, Rahe AJ. The Air Force Health Study: an epidemiologic investigation of health effects in Air Force Personnel following exposure to herbicides: reproductive outcomes. *Chemosphere.* 1992;25(1-2):217-218.

Yen S, MacMahon B. Genetics of anencephaly and spina bifida? *Lancet.* 1968;2:623-626.

Yerushalmy J. The relationship of parents' cigarette smoking to outcome of pregnancy: implications as to the problem of inferring causation from observed associations. *Am J Epidemiol.* 1971;93:443-456.

RESPIRATORY DISEASE

Amundson DE. Etiology of pneumonia in American naval recruits. *Am J Resp Dis.* 1991;143:A496.

Arlander TR, Pierce WE, Edwards EA, Peckinpaugh RO, Miller LF. Epidemiology and prevention of acute respiratory disease in naval recruits: IV. An epidemiologic study of respiratory illness patterns in Navy and Marine Corps recruits. *Am J Public Health.* 1965;55:67-80.

Bardana EJ Jr, Montanaro A. Tight building syndrome. *Immunol Allergy Pract.* 1986;8:74-88.

Bar-Ziv J, Goldberg GM. Simple siliceous pneumonoconiosis in Negev bedouins. *Arch Environ Health.* 1974;29:121-126.

Blood CG, Griffith DK. Ship size as a factor in illness incidence among U.S. Navy vessels. *Mil Med.* 1990;155:310-314.

Breiman FR, Butler CJ, Tenover CF, et al. Emergence of drug-resistant pneumococcal infections in the United States. *JAMA.* 1994;271:1831-1835.

Brundage JF, Scott RM, Lednar WM, Smith DW, Miller RN. Building-associated risk of febrile acute respiratory diseases in army trainees. *JAMA.* 1988;259:2108-2112.

Calabia B, Gleaves C, Leggett R, Gilbert DN. Asymptomatic pharyngeal carriage of *Mycoplasma pneumoniae. Interscience Conference on Antimicrobial Agents and Chemotherapy.* 1992. Abstract 543.

Carrougher GJ. Inhalation injury. *AACN Clin Issues Crit Care Nurs.* 1993;4(2):367-377.

Couch RB. Viruses and indoor air pollution. *Bull NY Acad Med.* 1981;57:907-921.

Dagan R, Ferne M, Sheinis M, et al. An epidemic of penicillin-tolerant group A streptococcal pharyngitis in children living in a closed community: mass treatment with erythromycin. *J Infect Dis.* 1987;156:514-516.

Dixon RE. Economic cost of respiratory tract infections in the United States. *Am J Med.* 1985;78(suppl):45-51.

Finland M. Recent advances in the epidemiology of pneumococcal infections. *Medicine.* 1942;21:307-344.

Garibaldi RA. Epidemiology of community-acquired respiratory tract infections in adults: incidence, etiology, and impact. *Am J Med.* 1985;78(suppl):32-37.

Grahn E, Holm E, Roos K. Penicillin tolerance in beta-streptococci isolated from patients with tonsillitis. *Scand J Infect Dis.* 1987;19:421-426.

Gray GC. Acute respiratory disease in the military. *Federal Practicioner.* 1995;January:27-33.

Gray GC, Duffy L, Paver R, et al. *Mycoplasma pneumoniae,* a frequent cause of pneumonias among US Marines. *Interscience Conference on Antimicrobial Agents and Chemotherapy.* 1994. Abstract 1264.

Gray GC, Escamilla J, Hyams, KC, et al. Hyperendemic *Streptococcus pyogenes* infection despite benzathine penicillin G prophylaxis. *N Engl J Med.* 1991;325:92-97.

Gray GC, Hyams KC, Wang SP, Grayston JT. *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* strain TWAR infections in US Marine Corps recruits. *Mil Med.* 1994;159:292-294.

Gray GC, Mitchell BS, Tueller JE, et al. Adult pneumonia hospitalizations in the US Navy: rates and risk factors for 6,522 admissions, 1981-1991. *Am J Epidemiol.* 1994;139:793-802.

Hicks JB. Tight building syndrome: when work makes you sick. *Occup Health Saf.* 1984;53:51-56.

Hines FJ. A comparison of clinical diagnoses among male and female soldiers deployed during the Persian Gulf War. *Mil Med.* 1993;158:99-101.

Respiratory Disease

Hinkle LE Jr, Murray SH. The importance of the quality of indoor air. *Bull NY Acad Med.* 1981;57:827-844.

Hirsch M, Bar-Ziv J, Lehmann E, Goldberg GM. Simple siliceous pneumonoconiosis of bedouin females in the Negev Desert. *Clin Radiol.* 1974;25:507-510.

Hoeffler DF. Current patterns of acute respiratory disease in the United States Navy and Marine Corps. *Yale J Biol Med.* 1975;48:171-178.

Hoeffler DF, Melton LJ. Changes in the distribution of Navy and Marine Corps casualties from World War I through the Vietnam conflict. *Mil Med.* 1981;146:776-779.

Holmes KK, Miller FL, Edwards EA, Johnson DW. Etiology of pneumonia in nonrecruit military personnel. *Am J Med Sci.* 1971;260:264-269.

Kaplan EL. The group A streptococcal upper respiratory tract carrier state: an enigma. *J Pediatr.* 1980;97:337-345.

Kilburn KH, Lilis R, Holstein E. Silicosis. In: Last JM, ed. *Maxcy-Rosenau Public Health and Preventive Medicine.* New York, Ny: Appleton-Century-Crofts; 1980:598-644.

Kim SK, Kaplan LE. Association of penicillin tolerance with failure to eradicate group A streptococci from patients with pharyngitis. *J Pediatr.* 1985;107:681-684.

Klontz KC, Hynes NA, Gunn RA, Wilder MH, Harmon MW, Kendal AP. An outbreak of influenza A/Taiwan/1/86 (H1N1) infections at a naval base and its association with airplane travel. *Am J Epidemiol.* 1989;129:341-348.

Linenger MJ, Flinn S, Thomas B, Weech-Johnson C. Musculoskeletal and medical morbidity associates with rigorous physical training. *Clin J Sport Med.* 3:229-234.

Miller LF. Acute respiratory infections in naval personnel. *Mil Med.* 1964;129:526-532.

Miller LF, Rytel M, Pierce WE, Rosenbaum MJ. Epidemiology of nonbacterial pneumonia among naval recruits. *JAMA.* 1963;185:92-99.

Paparello SF, Bourgeois AL, Garst P, Hyams KC. Diarrheal and respiratory disease aboard the hospital ship, USNS Mercy T-AH 19, during Operation Desert Shield. *Mil Med.* 1993;158:392-395.

Pazzaglia G, Pasternack M. Recent trends of pneumonia morbidity in U.S. naval personnel. *Mil Med.* 1983;148:647-651.

Pazzaglia G, Walker RI. A retrospective survey of enteric infections in active-duty Mavy and Marine Corps personnel. *Mil Med.* 1982;147:27-33.

Policard A, Collet A. Deposition of siliceous dust in the lungs of the inhabitants of the Saharan regions. *Arch Ind Hyg Occup Med.* 1952;5:527-534.

Reichler M, Reynolds R, Schwartz B, et al. Epidemic of pneumococcal pneumonia at a military training camp. *Interscience Conference on Antimicrobial Agents and Chemotherapy.* 1991. Abstract 49.

Richards AL, Hyams KC, Watts DM, Rozmajzl PJ, Woody JN, Merrell BR. Respiratory disease among military personnel in Saudi Arabia during Operation Desert Shield. *Am J Public Health.* 1993;83:1326-1329.

Rosenbaum MJ, Edwards EA, Frank PF, Pierce WE, Crawford YE, Miller LF. Epidemiology and prevention of acute respiratory disease in naval recruits: I. Ten years' experience with microbial agents isolated from naval recruits with acute respiratory disease. *Am J Public Health.* 1965;55:38-46.

Rosenbaum MJ, Edwards EA, Hoeffler DF. Recent experience with live adenovirus vaccines in Navy recruits. *Mil Med.* 1975;140:251-257.

Schiott CR, Engbaek HC, Vergmann B, Al-Motez M, Kassim I. Incidence of drug resistance amongst isolates of *Mycobacterium tuberculosis* recovered in the Gizan area, Saudi Arabia. *Saudi Med J.* 1985;6:375-378.

Schwoeble AJ, Dalley AM, Henderson BC, Casuccio GS. Computer-controlled SEM and microimaging of fine particles. *J Metals.* 1988;40:11-14.

Seaton A, Legge JS, Henderson J, Kerr KM. Accelerated silicosis in Scottish stonemasons. *Lancet.* 1991;337:341-344.

Seppala H, Nissinen A, Jarvinen H, et al. Resistance to erythromycin in group A streptococci. *N Engl J Med.* 1992;5:292-297.

Sinclair DG, Hepburn NC, Bowen J, Winfield CR. Community-acquired pneumonia in the Gulf. *J R Army Med Corps.* 1991;137:126-127.

Stevens DL, Tanner MH, Winship J, et al. Severe group A streptococcal infection associated with a toxic shock-like syndrome and scarlet fever toxin A. *N Engl J Med.* 1989;321:1-7.

Stille WT, Pierce W, Crawford EY. Multiple infections in acute respiratory illness. Severity of illness of Naval recruits and independence of infectious agents. *J Infect Dis.* 1961;109:158-165.

Thomas RJ, Conwill DE, Morton DE, et al. Penicillin prophylaxis for streptococcal infections in the United States Navy and Marine Corps recruit camps, 1951-1985. *Rev Infect Dis.* 1988;10:125-130.

Thomas TL, Stewart PA. Mortality from lung cancer and respiratory disease among pottery workers exposed to silica and talc. *Am J Epidemiol.* 1987;125:35-43.

Top FH Jr. Control of adenovirus acute respiratory disease in U.S. Army trainees. *Yale J Biol Med.* 1975;48:185-195.

Vallyathan V, Shi X, Dalal NS, Irr W, Castranova V. Generation of free radicals from freshly fractured silica dust: potential role in acute silica-induced lung injury. *Am Rev Respir Dis.* 1988;138:1213-1219.

Wale MCJ. Influenza B, Q fever, and the consequences of febrile illness occurring during jungle warfare training: a clinical and serological study. *J R Nav Med Serv.* 1989;75:13-18.

SMOKE EFFECTS

Ainslie G. Inhalational injuries produced by smoke and nitrogen dioxide. *Respir Med.* 1993;87(3):169-174.

Al-Yakoob SN, Abdal Y, Nasrallah H, Al-Majed N. Exposure to particle-bound polyaromatic hydrocarbons in the Al-Mansoria residential area during the Kuwait oil fires: a qualitative appraisal of the absorption role. *Environ Int.* 1993;19(4):319-331.

American Academy of Arts and Sciences. *Kuwait Oil Fires Conference, draft final report.* Cambridge, Ma: September 26, 1991.

Bakan AS, Chlond A, Cubasch U, et al. Climate response to smoke from the burning oil wells in Kuwait. *Nature.* 1991;351(6325):367-370.

Baraka ME. A foresight: the burned oil wells in Kuwait [letter]. *Arch Otolaryngol Head Neck Surg.* 1992;118(6):663.

Browning KA, Allam RJ, Ballard SP, et al. Environmental effects from burning oil wells in Kuwait. *Nature.* 1991;351(6325):363-367.

Clark WR Jr. Smoke inhalation: diagnosis and treatment. *World J Surg.* 1992;16(1):24-29.

Coombe MD, Drysdale SF. Assessment of the effects of atmospheric oil pollution in post-war Kuwait. *J R Army Med Corps.* 1993;139(3):95-97.

Crebelli R, Fuselli S, Meneguz A, et al. *In vitro* and *in vivo* mutagenicity studies with airborne particulate extracts. *Mutat Res.* 1988;204:565-575.

Crespi CL, Liber HL, Behymer TD, Hites RA, Thilly WG. A human cell line sensitive to mutation by particle-borne chemicals. *Mutat Res.* 1985;157:71-75.

Crespi CL, Thilly WG. Mutagenesis in diploid human lymphoblasts competent for xenobiotic metabolism. *Mutat Res.* 1984;128:221-230.

Danish S, Madany IM. Concentrations of nitrogen dioxide throughout the state of Bahrain. *Environ Pollut.* 1992;77(1):71-78.

Darcey DJ, Everson RB, Putman KL, Randerath K. DNA adducts and exposure to burning oil [letter]. *Lancet.* 1992;339(8791):489.

Dech SW, Glaser R. Burning oil wells in Kuwait: smoke plue monitoring and effects on vegetation derived from AVHRR data. *Int J Remote Sens.* 1992;13(17):3243-3249.

DeFlora S, Bagnasco M, Izzotti A, D-agostini F, Pala M, Valerio F. Mutagenicity of polycyclic hydrocarbon fractions extracted from urban air particles. *Mutat Res.* 1989;224:305-318.

Department of Commerce. *The Kuwait Oil Fires - Air Quality Data and Reports.* Washington, DC: Gulf Program Office, NOAA; February 1992.

de Ratt WK. Polycyclic aromatic hydrocarbons and mutagens in ambient air particles. *Toxicol Environ Chem.* 1988;165:259-279.

de Ratt WK, Kooijman SALM, Gielen JWJ. Concentrations of polycyclic hydrocarbons in airborne particles in the Nederland and their correlation with mutagenicity. *Sci Total Environ.* 1989;66:95-114.

El-Shobokshy MS, Al-Tamrah SA, Hussain FM. Inhalable particulates and meteorological characteristics of the city of Riyadh, Saudi Arabia. *Atmos Environ.* 1990;24B:261-265.

EPA (Environmental Protection Agency). *Kuwait Oil Fires: Interagency Interim Report.* Washington, DC: EPA; 1991.

Smoke Effects

Friedman GK. Health experience of fire fighters. Presented at the National Institutes of Health Technology Assessment Workshop on the Persian Gulf Experience and Health; Bethesda, MD: April 27, 1994.

Hahn J. Environmental effects of the Kuwait oil field fires. *Environ Sci Technol.* 1991;25(9):1530-1532.

Haponik EF. Clinical smoke inhalation injury: pulmonary effects. *Occup Med.* 1993;8(3):430-468.

Hobbs PV, Radke LF. Airborne studies of the smoke from the Kuwait oil fires. *Science.* 1992;256(5059):987-991.

Horgan J. The danger from Kuwait's air pollution [news]. *Sci Am.* 1991;265(4):30.

Horgan J. Up in flames - Kuwait's burning oil wells are a sad test of theories. *Sci Am.* 1991;264:7-9.

Horgan J. Burning questions - scientists launch studies of Kuwait's oil fires. *Sci Am.* 1991;265:17-24.

Husain T, Khan SM. Air monitoring network program in Saudi Arabia. *Sci Total Environ.* 1982;41-46.

Johnson DW, Kilsby CG, McKenna DS, et al. Airborne observations of the physical and chemical characteristics of the Kuwait oil smoke plume. *Nature.* 1991;353(6345):617-621.

Kelsey KT, Xia F, Bodell WJ, et al. Genotoxicity to human cells induced by air particulates isolated during the Kuwait oil fires. *Environ Res.* 1994;64(1):18-25.

Kennedy JM. Smoke billows from Wafra oil field torched by Iraqi troops [AP Photo story]. *LA Times.* 1991;January 24:A7.

Kennedy JM, Healy M. 150 wells set ablaze. *LA Times.* 1991; February 23:A1.

Khattack MN. Natural and anthropogenic atmospheric particulates at UPM campus, Dhahran, Saudi Arabia. *J Air Pollut Control Assoc.* 1982;32:1153-1155.

LA Times. Black rain on Bushehr Province of Iran. *LA Times.* 1991;January 24:A7.

Lewtas J. Genotoxicity of complex mixtures: strategies for the identification and comparative assessment of airborne mutagens and carcinogens from combustion sources. *Fundam Appl Toxicol.* 1988;10:571-589.

Meteorology and Environmental Protection Administration. *Environmental Impact of Oil Burning in the Kuwaiti Oilfields: A Preliminary Evaluation.* Geneva: Expert Meeting on the Atmospheric Part of the Emergency Response to the Kuwait Oilfield Fires: The World Meteorological Organization; April 27-30, 1991.

Moller M, Alfheim I, Larssen S, Mikalsen A. Mutagenicity of airborne particles in relation to traffic and air pollution parameters. *Environ Sci Technol.* 1982;16:221-225.

Mulholland G, Benner B, Fletcher R, Steel E, Wise S, May W. *Analysis of Some Sample from Oil Well Fires in Kuwait.* Gaithersburg, Md: Report Submitted to US Department of Commerce, NOAA, Report to Test FR 3985; 1991.

Nasralla MM. Air pollution in the semitropical Saudi urban area. *Environ Int.* 1983;9:255-264.

Orzel RA. Toxicological aspects of fire smoke: polymer pyrolysis and combustion. *Occup Med.* 1993;8(3):414-429.

Porter HO. Aviators intoxicated by inhalation JP-5 fuel vapors. *Aviat Space Environ Med.* 1990;61(7):654-656.

Raveendran E, Al-Mahmood AM. Trace metals in the atmosphere of Bahrain during the Kuwait oil fires. *Environ Technol.* 1992;13(11):1097-1100.

Rayman RB, McNaughton GB. Smoke/fumes in the cockpit. *Aviat Space Environ Med.* 1983;54(8):738-740.

Readman JW, Fowler SW, Villeneuve UP, Cattini C, Oregioni B, Mee LD. Oil and combustion-product contamination of the Gulf marine environment following the war. *Nature.* 1992;358(6388)662-665.

Renner MG. Military victory, ecological defeat. *Environ Prof.* 1991;13(3):285-288.

Riley JJ, Hicks NG, Thompson TL. Effect of Kuwait oil field fires on human comfort and environment in Jubail, Saudi Arabia. *Int J Biometeorol.* 1992;36(1):36-38.

Rosenwicz B. Iraqis set fire to oil sites in Kuwait. *Wall St J.* 1991;January 23: A3.

Rowe DR, Al-Dhowalia KH, Mansour ME. Indoor-outdoor nitric oxide and nitrogen dioxide concentrations at three sites in Riyadh, Saudi Arabia. *J Air Waste Manage Assoc.* 1991;41(7):973-976.

Shusterman DJ. Clinical smoke inhalation injury: systemic effects. *Occup Med.* 1993;8(3):469-503.

Simons M. British study disputes lengthy climatic role for Kuwait oil fires. *NY Times.* 1991;April 16: C4(L).

Small RD. Environmental impacts of fires in Kuwait. *Nature.* 1991;350:11-12.

Spengler J. *"Harvard School of Public Health's Kuwait Oil First Investigations"* [Abstract]. Cambridge, Ma: Kuwait Oil Fires Conference; August 12-14, 1991.

Stevens R, Pinto J, Mamane Y, et al. Chemical and physical properties of emissions from Kuwaiti oil fires. *Water Sci Technol.* 1993;27(7-8):223-233.

Suess MJ. The environmental load and cycle of polycyclic aromatic hydrocarbons. *Sci Total Environ.* 1976;6:239-250.

Tokiwa H, Kitamori S, Horikawa K, Nakagawa R. Some findings on the mutagenicity in airborne particulate pollutants. *Environ Mutagen.* 1983;5:87-100.

Tupper CR. Chemical hazards in aeromedical aircraft. *Aviat Space Environ Med.* 1989;60(1):73-75.

United Nations. *Kuwait.* New York, Ny: United Nations Department of Public Health, DPI/1157-40831; September 1991.

U.S. Army Environmental Hygiene Agency (USAEHA). *Interim Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91, 5 May - 15 September 1991.* Aberdeen Proving Ground, Md: USAEHA; 1992.

USAEHA. Final Report - *Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91, 5 May - 3 December 1991.* Aberdeen Proving Ground, Md: USAEHA; 1994.

USAEHA. *Biological Surveillance Initiative. Appendix F of Final Report Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91, 5 May - 3 December 1991.* Aberdeen Proving Ground, Md: USAEHA; 1994.

U.S. interagency team proposes program to quantify effects of Kuwait oil fires [news]. *J Air Waste Manage Assoc.* 1991;41(6):852-853.

Zhukov GA. The consequences of the war in the Persian Gulf region (a review of the literature). *Voen Med Zh.* 1992;(12):17-19.

Zonn IS. The Gulf War and the problem of desertification. *Probl Osvo Pustyn.* 1991;(3-4):133-144.

WAR AND DISEASE

Abramowicz M. Health problems in the Persian Gulf. *The Medical Letter.* 1991;33:13-15.

Adley R. Naval Party 1036: its role in the Gulf conflict. *J R Nav Med Serv.* 1992;78(2):65-71.

Ankney B. Iraq possessed large biological research program, U.N. team says. *US Med.* 1992;28:3,40-41.

Anonymous. Gulf illness: resolution elusive. *US Med.* 1994;30:3, 14.

Baker MS, Strunk HK. *Diving Hazards in the Persian Gulf: Proceedings of the U.S. Naval Institute,* 116/7/1049. 1990:74-76.

Beale P. Gulf illness [letter]. *BMJ.* 1994;308:1574.

Bedard P. Panel created to probe Gulf illness. *Washington Times.* May 27, 1995.

Berezuk GP, McCarty GE. Investigational drugs and vaccines fielded in support of Operation Desert Storm. *Mil Med.* 1992;157:404-406.

Blackwell J. *Thunder in the Desert:The Strategy and Tactics of the Gulf War.* New York, Ny: Bantam Books; 1991.

Blanck RR, Bell WH. Medical support for American troops in the Persian Gulf. *N Engl J Med.* 1991;324(12):857-859.

War and Disease

Blanck RR, Bell WH. Special reports: medical aspects of the Persian Gulf war. *N Engl J Med.* 1991;324:857-859.

Borkan J, Reis S. Untoward effects of gas masks during the Persian Gulf war. *N Engl J Med.* 1991;325:582-583.

Borkan J, Shvartzman P, Reis S, Morris AG. Stories from the sealed rooms: patient interviews during the Gulf War. *Fam Pract.* 1993;10(2):188-192.

Brown D. Diagnosis unknown: Gulf War syndrome. *The Washington Post.* 1994;July 24:

Choo V. UK Gulf War syndrome assessment programme deemed appropriate. *Lancet.* 1995;346:434.

Clinton to create a panel to look into Gulf War illness. *The New York Times.* March 7, 1995.

Clinton vows aid for sick Gulf vets. Focus on research, care wins applause. *Washington Times.* March 7, 1995.

Clinton woos lukewarm veterans. VFW applaud vow to fight VA cuts, study 'Gulf War Syndrome'. *The Washington Post.* March 7, 1995.

Committee on the DoD Gulf syndrome. *Comprehensive Clinical Evaluation Program: First Report.* 1994.

Committee on Veterans' Affairs. *Is Military Research Hazardous to Veterans' Health? Lessons Spanning Half a Century;* 1994.

Committee to Review the Health Consequences of Service During the Persian Gulf War. *Health Consequences of Service During the Persian Gulf War: Initial Findings and Recommendations for Immediate Action.* Washington, DC: National Academy Press; 1995.

Cotton P. Veterans seeking answers to syndrome suspect they were goats in Gulf War. *JAMA.* 1994;251:1559-1560, 1561.

Coyne J, Kessler RC, Tal M, Turnbull J, Wortman CB, Greden JF. Living with a depressed person. *J Consult Clin Psychol.* 1987;55:347-352.

CRS (Congressional Research Service). *Iraq-Kuwait Crisis: A Chronology of Events, July 17, 1990 - December 23, 1991.* Washington, D.C.: The Library of Congress; 1992.

Daniell FD, Walz SE, Crafton LD, Bolton HT. Field preventive medicine and epidemiological surveillance: the Beirut, Lebanon experience. *Mil Med.* 1985;150:171-176.

Danon YL, Shemer J, eds. The Persian Gulf war: Israeli medical experience and perspectives. *Is J Med Sci.* 1991;27:601-704.

Daxon EG. *Protocol for Monitoring Gulf War Veterans With Imbedded Fragments of Depleted Uranium.* AFRRI Technical Report 93-2. Bethesda, MD: Armed Forced Radiobiology Research Institute: 1993.

Defense Science Board. *Final Report: Defense Science Board Task Force on Persian Gulf War Health Effects.* Washington, D.C.: Office of the Under Secretary of Defense for Acquisition and Technology; 1994.

Department of Defense Report. *Clinical Evaluation Program for Gulf War Veterans: Preliminary Status Report on the First 1,000 Patients: 1994.*

Department of Defense Report. *Clinical Evaluation Program for Gulf War Veterans: Second Interim Report on 2,076 Participants.*

Department of Defense Report. *Comprehensive Clinical Evaluation Program (CCEP) for Gulf War Veterans: Report on 10,020 Participants.* August 1995.

Department of Veterans Affairs. *Update on Persian Gulf Research.* Department of Veterans Affairs, Office of Research and Development, Medical Research Service; 1994.

Dreze J, Ghazdar H. *Hunger and Poverty in Iraq. Vol. 32 of Wider Series.* London: London School of Economics; 1991:1-63.

Dunnigan JF, Bay A. *From Shield to Storm: High-Tech Weapons, Military Strategy, and Coalition Warfare in the Persian Gulf.* New York, Ny: William Morrow; 1992.

Dyer C. Gulf veteran gets pension for desert fever. *Biol Med.* 1994;309:1961.

Eusol recommendation for Gulf War wounds criticized [letter]. *Nurs Times.* 1991;87(8):14.

Farrar JT. *Under Secretary for Health Information Letter. Medical Care Programs for Persian Gulf Veterans, Including Comprehensive Clinical Examination Protocol.* Washington, DC; 1994.

Fedele K. The Seabees go to war. *Naval Civil Engineer.* 1991;Summer: 3-13.

Ficarra BJ. Disputanda. Medical mystery: Gulf War syndrome. *J Med.* 1995;26(1,2):87-94.

Flanders L. A lingering sickness. *The Nation.* 1995;January 23:94, 96.

France D. The families who are dying for our country. *Redbook.* 1994; Sept: 114-117, 147-148.

Furst A. The effects of the Gulf War in Israel - report from a rural family medical practice. *Public Health Rev.* 1992-1993;20(3-4):307-312.

Fumento, M. What is Gulf War syndrome? *American Spectator.* 1995:28-34.

Garfield RM, Neugut AI. Epidemiologic analysis of warfare: a historical review. *JAMA.* 1991;266:688-692.

Garvey BT. "Knock, knock": Social responses to the Gulf War. *Contemporary Social Psychology.* 1991;15:161-165.

Gasser RA Jr, Magill AJ, Oster CN, Tramont EC. The threat of infectious disease in Americans returning from Operation Desert Storm. *N Engl J Med.* 1991;324:859-864.

Geller JL, White C, Fisher W, Sorgi P, Jarvey AM. State hospital patients' views about Operation Desert Storm. *Hosp Comm Psychiatry.* 1992;43(8):833-835.

Golander H, Ehrenfeld M, Bergman R. The Gulf War: reaction and action in institutions for the aged in Israel. *J Adv Nurs.* 1992;17(10):1156-1165.

Goldberg J, Eisen SA, True WR, Henderson WG. Health effects of military service. Lessons learned from the Vietnam experience. *Ann Epidemiol.* 1992;2(6):841-853.

Goodman NW. Gulf war syndrome and other media 'causes.' *British Journal of Hospital Medicine.* 1995;54(1):46.

Greene S. Push for Gulf syndrome research. *Nature.* 1993;364:659.

Gulf War Symptoms remain puzzling. *JAMA.* 1992;268:2619.

Gunby P, Merwick C. Military medical equipment, techniques often required years of preparation. *JAMA.* 1991;265:1797.

Helmkamp JC. Epidemiological characteristics of U.S. fatalities during Desert Storm. *Mil Med.* 1992;157:A7.

Helmkamp JC. United States military casualty comparisons during the Persian Gulf War. *J Occup Med.* 1994;36:609-615.

Hines JF. Ambulatory health care needs of women deployed with a heavy armor division during the Persian Gulf war. *Mil Med.* 1992;157:219-221.

Hines JF. A comparison of clinical diagnoses among male and female soldiers deployed during the Persian Gulf war. *Mil Med.* 1993;158:99-101.

Hiss J, Arensburg B. Suffocation from misuse of gas masks during the Gulf War. *BMJ.* 1992;304:92.

Hyams KC, Bourgeois AL, Escamilla J, Burans J, Woody JN. The Navy forward laboratory during Operations Desert Shield/Desert Storm. *Mil Med.* 1993;158:729-732.

Institute of Medicine. *Health Consequences of Service During the Persian Gulf War: Initial Findings and Recommendations for Immediate Action.* Washington, DC: National Academy Press; 1995.

Iraq: Immunization, Diarrhoeal Diseases. Maternal and Childhood Mortality Survey. Evaluation Series No. 9. Amman, Jordan: UNICEF Regional Office for the Middle East and North Africa; 1990.

Kang HK, Dalager NA, Lee, KY. *Health Surveillance of Persian Gulf War Veterans: a Review of the Department of Veterans Affairs Persian Gulf Registry and In-Patient Treatment Files.* Environmental Epidemiology Service, Department of Veterans Affairs; June 1995.

Kelley R, Hycke K. Medical threat assessment: Saudi Arabia, Kuwait, Iraq. *WRAIR Communicable Disease Report.* 1990;3:3-8.

Keogh JP. *Proposal to the Department of Veterans Affairs Program for Follow-up Monitoring of Gulf War Veterans with Imbedded Fragments of Depleted Uranium.* Baltimore, Md: Baltimore Veterans Affairs Medical Center; 1993.

Kizer KW. The health of Persian Gulf veterans [letter]. *JAMA.* 1995;273(23):1817.

Knudson GB. Operation Desert Shield: medical aspects of weapons of mass destruction. *Mil Med.* 1991;156:267-271.

Korenyi-Both AL, Molnar AC, Korenyi-Both AL, Fidelus-Gort RF. Al Eskan disease: Desert Storm pneumonitis. *Mil Med.* 1992;157:452-462.

Lancet News in Brief. Gulf War syndrome. *Lancet.* 1995;345.

Lancet News in Brief. No news for sick Desert Storm veterans. *Lancet.* 1995;345.

Ledford FF. Medical support for Operation Desert Storm. *Journal of the U.S. Army Medical Department.* 1992;8:3-6.

Lee YT, Alvarez JD, Wilson JA, Harned LB. Operation Desert Storm: clinical experiences at the 13th Evacuation Hospital, a Wisconsin National Guard Unit. *Wis Med J.* 1992;91(8):480-482.

Lehman DR. Continuing the tradition of research on war: the Persian Gulf war. *Journal of Social Issues.* 1993;49:1-13.

Levy A. Compensation neurosis rides again. *Brain Injury.* 1992 Sep-Oct;6(5):401-10.

Lie D. `No longer nineteen'. A review of Vietnam veterans in everyday practice. *Aust Fam Physician.* 1992;21(6):759-764.

Marple R, Lucey C, Kroenke A, et al. A prospective study of concerns and expectations in patients presenting with common symptoms; and the 2 week outcome in patients presenting with common physical complaints. *Abstracts in Clinical Research.* 1993;41:579A.

McCarthy M. US study finds no Gulf War syndrome. *Lancet.* 1995;346:434.

Medecins Sans Frontieres. Kurdistan: helping an abandoned people. *Alert.* Fall-Winter, 1991-1992:1-4.

Medical care in the Persian Gulf War. Washington, DC. *US Medicine.* 1991.

Medical News and Perspectives. Institute of Medicine calls for coordinated studies of Gulf War veterans' health complaints. *JAMA.* 1995;273(6):444-445.

Medicine in the Gulf War. *US Med.* 1991;27:1-113.

Miller G. Does war stress contribute to hypertension? *Aust Fam Physician.* 1993;22(5):707-710.

Milner IB, Axelrod BN, Pasquantonio J, Sillanpaa M. Is there a Gulf War syndrome? [letter] *JAMA.* 1994;271:661.

Morgan D. Gulf vets have more ailments, study finds. *Washington Post.* June 16, 1995.

Murray-Leisure KA, Daniels MO. *Visceral and Skin Aspect of "Persian Gulf Mystery Disease"* [abstract no. J159]. Program and abstracts of the 34th Interscience Conference on Antimicrobial Agents and Chemotherapy (Orlando, FL). Washington, DC: American Society for Microbiology; 1994.

National Child Health Survey. Baghdad, Iraq: Ministry of Health; 1990.

National Institutes of Health Technology Assessment Workshop Panel. The Persian Gulf experience and health. *JAMA.* 1994;272:391-396.

Nelson SS. Soldiers will have to fight heat, dust, disease. *Army Times.* 1990;Aug 20:16.

Nelson SS. Gulf vets' claims stymied. *Army Times.* June 5, 1995.

Nelson SS. Panel probes Gulf syndrome effort. *Army Times.* June 19, 1995.

Nelson SS. Indiana's reservists still sick, unhappy. *Army Times.* June 19, 1995.

Nelson SS. Gulf war papers, coming soon to computer. *Navy Times.* August 7, 1995.

Nicholson NL. Massive Cover Up of Desert Storm Illness Source. *Criminal Politics.* 1995;March:32-36.

O'Ballance E. *The Gulf War.* London, England: Brassey Defense Publishers. 1988;103/104:182-183.

O'Donnell FL. *Environmental Conditions in Saudi Arabia.* Presentation to NIH Technology Assessment Workshop Panel on the Persian Gulf Experience and Health. Bethesda, Md; 1994.

Oster CN, Sanford JP. Febrile illness in a Desert Storm veteran. *Hosp Pract.* 1992;27:145-148, 151, 155-160.

War and Disease

Painful sex joins Gulf syndrome symptoms. *The Oregonian.* March 1, 1995.

Palinkas LA, Coben P. Disease and non-battle injuries among U.S. Marines in Vietnam. *Mil Med.* 1988;153(3):150-155.

Panel urges further research on Gulf War illness, possible increased cancer risks in veterans. *J Natl Cancer Inst.* 1994;86:820-822.

Persian Gulf Veterans Coordinating Board. *Summary of the Issues Impacting Upon the Health of Persian Gulf Veterans, Version 3.0.* Washington, DC: Support Office of the Persian Gulf Veterans Coordinating Board; 1994.

Persian Gulf Veterans Coordinating Board. Unexplained illnesses among Desert Storm veterans: a search for causes, treatment, and cooperation. *Arch Intern Med.* 1995;155:262-268.

Pine A. Government Study of Veterans Finds No Evidence of a 'Gulf War Disease.' *The Los Angeles Times.* 1995;August 2.

Public health consequences of acute displacement of Iraqi citizens — March-May 1991. *MMWR.*1991;40:443-446.

Quin NE, Royal Army Medical Corps. The impact of diseases on military operations in the Persian Gulf. *Mil Med.* 1982;147:728-734.

Report of the Defense Science Board Task Force on Persian Gulf War Health Effects. Washington, DC: Office of the Under Secretary of Defense for Acquisition and Technology; 1994.

Report to Congress, United States Gulf Environmental Technical Assistance. Washington, DC: Gulf Task Force, US Environmental Protection Agency; 1991.

Richards AL, Hyams KC, Merrell BR, et al. Medical aspects of Operation Desert Storm [letter]. *N Engl J Med.* 1991;325:970.

Richter ED. Fewer injuries but more deaths from road accidents during the Persian Gulf war. *Isr J Med Sci.* 1991;27(11-12):631-635.

Riley EB. *Operation Desert Shield/Desert Storm. History of Participation by the U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, Maryland (7 August 1990 - 31 December 1991).* Aberdeen Proving Ground, Md: USAEHA; 1992.

Roy MJ, Chung RCY, Huntley DE, Blanck RR. Evaluating the symptoms of Persian Gulf War veterans. *Fed Pract.* 1994;11:13-6,22.

Sanford JP. *Gulf War Syndrome: Proposed Provisional Case Definition.* Memo to Major General Ronald Blanck from J. P. Sanford, M.D.: 1994.

Sato N, Obeid O. Brun T. Malnutrition in southern Iraq. *Lancet.* 1991;338:1202.

Scurfield RM. The collusion of sanitization and silence about war: aftermath of "Operation Desert Storm". *Journal of Traumatic Stress.* 1992;5(3):505-512.

Secord E, Claude S. Newton C. War souvenir poisoning. *Am J Dis Child.* 1991;1445:724.

Secretary of Veterans Affairs. *Persian Gulf Veterans Research Activity Report.* March 1995.

Spencer L. Colonel Alm recounts vital role of veterinarians in Persian Gulf conflict. *J Am Vet Med Assoc.* 1991;199:305-309.

Stanton MD, Figley CR. Treating the Vietnam veteran within the family system. In: Figley CR, ed. *Stress Disorders Among Vietnam Veterans.* New York, Ny: Brunner/Mazel; 1978:281-295.

Stires LK. The Gulf "War" as a sanctioned massacre. *Contemporary Social Psychology.* 1991;15:139-143.

Stong GC, Hope JW, Kalenian MH. Medical evacuation experience of two 7th Corps medical companies supporting Desert Shield/Desert Storm. *Mil Med.* 1993;158:108-13.

Stretch RH, Bliese PD, Marlowe DH, Wright KM, Knudson KH, Hoover CH. Physical health symptomatology of Gulf War-era service personnel from the states of Pennsylvania and Hawaii. *Mil Med.* 1995;160:131-136.

The Harvard Study Team. The effect of the Gulf crisis on the children of Iraq. *N Engl J Med.* 1991;325:977-980.

Tolsdorf CC. Social networks, support, and coping: an exploratory study. *Fam Process.* 1976;15:407-417.

Torok PG, Roley E, Davis DL. Ophthalmomyiasis during Operation Desert Shield. *Mil Med.* 1991;156:438-439.

USA Today. Gulf vets deserve better than brushoff from brass. *USA Today.* August 17, 1995:10.

War and Disease

U.S. Army Medical Research, Development, Acquisition and Logistics Command (Provisional). *The General Well-Being of Gulf War Era Service Personnal From the States of Pennsylvania and Hawaii: A Survey*. Arlington, Va: Office of the Assistant Secretary of Defense - Health Affairs, the Pentagon; 1994.

U.S. Department of Health and Human Services. Unexplained illness among Persian Gulf veterans in an air National Guard Unit: preliminary report - August 1990-March 1995. *MMWR*. 1995;44(23):443-447.

VA. *Returning Persian Gulf Troops: First Year Findings*. West Haven, Ct: VA Northeast Program Evaluation Center; 1992.

VA. *Update on Persian Gulf Research*. Prepared by the Department of Veterans Affairs, Office of Research and Development, Medical Research Service. In collaboration with the Persian Gulf Interagency Research Coordinating Council. Washington, DC: VA Central Office; 1994.

Whelan EM. No 'syndrome' exists. *USA Today*. August 17, 1995:10.

White CS, Adler WH, McGann VG. Repeated immunization: possible adverse effects: reevaluation of human subjects at 25 years. *Ann Intern Med*. 1974;81:594-600.

Williams CM. The veteran system - with a focus on woman partners: theoretical considerations, problems, and treatment strategies. In: Williams T, ed. *Post Traumatic Stress Disorders of Vietnam Veterans*. Cincinnati, Oh: Disabled American Veterans; 1980:73-124.

Writer JV, DeFraites RF, Brundage JF, Kelley PW. *Comparative Mortality Among US Military Personnel Worldwide During Operations Desert Shield and Desert Storm*. Washington, DC: Walter Reed Army Institute of Research; 1995.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704–0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATE COVERED
	AUGUST 1995	NEW May 94 – Aug 95

4. TITLE AND SUBTITLE

TOPICAL BIBLIOGRAPHY OF PUBLISHED WORKS REGARDING THE HEALTH OF VETERANS OF THE PERSIAN GULF WAR

5. FUNDING NUMBERS

Program Element: 63706N
Work Unit Number:
P4464.001 – 6423

6. AUTHOR(S)

CORNELISON, COLLEEN M; GRAY, GREGORY C

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(S)

Naval Health Research Center
P. O. Box 85122
San Diego, CA 92186–5122

8. PERFORMING ORGANIZATION

Technical Document 95–3C

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(S)

Naval Medical Research and Development Command
National Naval Medical Center
Building 1, Tower 2
Bethesda, MD 20889–5044

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION/AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

We have constructed a topical bibliography for the benefit of researchers conducting studies among Persian Gulf War veterans. The document was framed around a bibliography of the Persian Gulf War and associated topics that was prepared by Jacqueline Van de Kamp, M.L.S., Specialized Information Services, National Library of Medicine, and John H. Ferguson, M.D., Office of Medical Applications of Research, National Institutes of Health. Their document was generated in April of 1994 and contained 594 citations. Our document contains 1,751 references, organized alphabetically by first authors' last name, into the following categories: Anthrax, Cancer, Chemical Warfare, Chronic Fatigue Syndrome, Fibromyalgia, Gastrointestinal Disease, Insecticides, Leishmaniasis, Multiple Chemical Sensitivity, Other Infectious Disease, Other Psychiatric Disease, Other Toxins and Their Treatment, Posttraumatic Stress Disorder, Pyridostigmine, Q Fever, Reproductive Disease, Respiratory Disease, Smoke Effects, and War and Disease.

14. SUBJECT TERMS

Persian Gulf War, Anthrax, Cancer, Chemical Warfare, Chronic Fatigue Syndrome, Fibromyalgia, Gastrointestinal Disease, Insecticides, Multiple Chemical Sensitivity, Infectious Disease, Psychiatric Disease, Toxins and

15. NUMBER OF PAGES

74

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	Unlimited

NSN 7540–01–280–5500

Standard Form 298 (Rev. 2–89)
Prescribed by ANSI Std. Z39–18
298–102

14. SUBJECT TERMS (continued)

Their Treatment, Posttraumatic Stress Disorder, Pyridostigmine, Q Fever, Reproductive Disease, Respiratory Disease, Smoke Effects, War and Disease.

DEPARTMENT OF DEFENSE REPORT

COMPREHENSIVE CLINICAL EVALUATION PROGRAM (CCEP)

FOR GULF WAR VETERANS

Report on 10,020 Participants

AUGUST 1995

#567

ABSTRACT

In response to veterans' concerns about potential health effects resulting from service during Operations Desert Storm/Shield, the Department of Defense (DoD) initiated the Comprehensive Clinical Evaluation Program (CCEP). To date, the CCEP has provided in-depth medical examinations to approximately 13,000 service and family members entitled to DoD health care. This descriptive case series report summarizes the diagnostic results of 10,020 participants who have finished their medical evaluations. Designed as a clinical rather than a research program, the results of the CCEP provide insight into the nature of symptoms and diagnoses in this self-selected group of individuals. In general, the demographic characteristics of CCEP participants represent a cross-section of Persian Gulf War veterans as a group. CCEP participants self-report a range of wartime occupational and environmental exposures. Symptoms and diagnoses seen in CCEP participants resemble those seen in the general population and in patients seeking primary care. Psychological, musculoskeletal, and nonspecific conditions represent the major categories of primary diagnoses, and may occur more frequently in the CCEP than in other primary care settings. Research studies with comparison groups of non-deployed Persian Gulf-era veterans will clarify whether or not these conditions may be more common among Persian Gulf War veterans. Severe disability measured in terms of lost work days does not appear to be a major characteristic of the clinical profile of CCEP participants. However, participants experiencing disabling symptoms may benefit from programs which have been established at DoD Specialized Care Centers that focus on rehabilitation, restoration of function and promotion of general well being. Finally, based on the CCEP experience to date, there exists no clinical evidence for a new syndrome or unique illness among Persian Gulf veterans. The results of the CCEP are consistent with conclusions of a National Institutes of Health Technology Assessment Workshop that "no single disease or syndrome is apparent, but rather multiple illnesses with overlapping symptoms and causes." DoD will arrange for independent researchers to have access to the CCEP data in the future.

Table of Contents

Tables

Page

Figures

Page

EXECUTIVE SUMMARY

COMPREHENSIVE CLINICAL EVALUATION PROGRAM FOR GULF WAR VETERANS: REPORT ON 10,020 PARTICIPANTS

Approximately 697,000 U.S. service members deployed to the Persian Gulf in 1990/1991 for Operations Desert Shield/Desert Storm (ODS/S). The vast majority of troops returned from this large deployment healthy and remain fit for duty today. In response to Gulf War veterans' concerns about the potential health effects of service in ODS/S and to further investigate the nature of their illnesses, the Departments of Defense (DoD) and Veterans Affairs (DVA) developed similar, comprehensive clinical evaluation programs. The DoD's Comprehensive Clinical Evaluation Program (CCEP) provides an in-depth medical evaluation for DoD beneficiaries who are experiencing illnesses which may be related to their service in the Persian Gulf. Currently, the program has enrolled nearly 23,000 participants. Approximately 17,000 of these participants have requested an examination, of which over 13,000 have finished the evaluation process, and the records of 10,020 have been verified and entered into the CCEP database.

This descriptive case series report summarizes the diagnostic results of over 10,000 systematic clinical evaluations completed through the CCEP. The CCEP was designed primarily as a clinical rather than research program. Self-selection of patients, recall bias, inability to validate self-reported exposures and lack of a control group limit the relevance of CCEP findings to other Persian Gulf veterans. However, the large size of the CCEP cohort and the thoroughness of the CCEP examinations provide considerable clinical insight towards understanding the nature of these veterans' illnesses and health concerns. Ongoing and planned DoD/VA/HHS sponsored epidemiologic studies involving control/comparison populations will characterize further the health consequences of the Persian Gulf War. Based on the evaluation of 10,020 participants, our findings include:

- To date, the CCEP has identified no clinical evidence for a new or unique illness or syndrome among Persian Gulf veterans. The results of the CCEP are consistent with the conclusion of a National Institutes of Health Technology Assessment Workshop that "no single disease or syndrome is apparent, but rather multiple illnesses with overlapping symptoms and causes."

- Symptoms reported by CCEP participants are similar to those seen in patients seeking primary care based on studies of outpatient practice and of the general U.S. population. CCEP patients demonstrate a broad cross-section of diagnoses which would be expected in this large population.

- Generalized symptoms such as fatigue, joint pain, headache, and sleep disturbances are very common among CCEP participants. Published studies of patients with these types of generalized symptoms have shown that 20-75% of them lack a clear-cut or discrete physical explanation or "cause" after a thorough medical evaluation. Similarly, it is likely that some CCEP participants may also lack a discrete physical explanation for their generalized symptoms.

- The distribution of International Classification of Diseases-9th Revision, Clinical Modification (ICD-9-CM) diagnostic categories seen in CCEP participants resembles that seen in the general population and in patients seeking primary care. "Psychological," "Signs, Symptoms, Ill-Defined Conditions," and "Musculoskeletal & Connective Tissue" represent the major ICD-9-CM categories of primary diagnoses.

- Severe disability, measured in terms of reported lost work days, is not a major characteristic of CCEP participants. Most CCEP participants (81%) had not missed work because of illness or injury during the 90 days prior to their initial evaluation. Seven percent of CCEP participants self-reported missing more than one week of work due to illness.

- Comparisons of CCEP participants to patients in outpatient medical settings are limited because of differences in patient populations. However, preliminary conclusions are as follows:

    * The most common psychological conditions found in CCEP participants are: tension headache; nonspecific, mild or stress-related anxiety and/or depression; posttraumatic stress disorder (PTSD). The prevalence of psychological diagnoses among CCEP participants may be higher than that observed in other patients seen in general medical practice.

    * CCEP diagnoses include a group of well-defined conditions not classified elsewhere in the ICD-9-CM coding system (e.g. sleep apneas), generalized symptoms, abnormal laboratory tests, and nonspecific physical findings. These diagnoses which are categorized as "Signs, Symptoms and Ill-Defined Conditions" according to the ICD-9-CM coding system may be more common in the CCEP compared to patients seen in general medical practice.

    * Musculoskeletal and connective tissue diseases (joint pain, osteoarthritis, backache) are common diagnoses seen in CCEP participants. These conditions appear to occur more frequently in the CCEP population compared to patients seen in general medical practice.

- DoD will continue to provide comprehensive quality health care to eligible Persian Gulf veterans, and will maintain an ongoing search for unique symptom/illness patterns. The Department is committed to a continuing exchange of relevant information with other government agencies and Gulf War veterans to further understand this public health issue.

INTRODUCTION

Approximately 697,000 U.S. service members deployed to the Persian Gulf in 1990/1991 for Operations Desert Shield/Desert Storm (ODS/S). Medical readiness planning and preventive medicine measures taken by the DoD contributed to U.S. military forces experiencing the lowest disease non-battle injury (DNBI) rate of any major conflict. The vast majority of soldiers, sailors, airmen, and marines returned from this large deployment healthy and remain fit for duty today. Since ODS/S, veterans seeking medical care have had a wide range of conditions that would be expected in such a large adult population. Some service members have had persistent symptoms which they believe are related to their experience in the Persian Gulf War. In response to Gulf War veterans' concerns about their health following ODS/S, the Departments of Defense (DoD) and Veterans Affairs (VA) developed similar comprehensive clinical evaluation programs. To date, the DoD has enrolled approximately 23,000 participants eligible for DoD health care in the Comprehensive Clinical Evaluation Program (CCEP).

In December, 1994, the DoD issued its preliminary status report on the first 1,000 patients to complete the CCEP. Since that report, the Department has continued an aggressive outreach effort to provide evaluation and care to veterans who are experiencing symptoms or illnesses which they feel may be related to their service in the Persian Gulf. The DoD provided an update on March 10, 1995, regarding the results of 2,076 medical evaluations accomplished through the CCEP. This report summarizes program activities through May 31, 1995, and includes the clinical findings from 10,020 patients who have completed their CCEP evaluations.

Potential Health Risks Associated With Persian Gulf Deployment

In order to better understand the potential causes of illnesses and most effective treatments for Gulf War veterans, a thorough review of the potential health risks associated with service in the Persian Gulf is necessary. These risks include: physical and psychological stress, possible reactions to prophylactic drugs and vaccines, infectious diseases, and potential exposures to environmental hazards.[1]

Physical and psychological stressors were major characteristics of the Persian Gulf. The effect of both acute and chronic stress is a major etiologic consideration when evaluating Persian Gulf veterans. U.S. troops entered a bleak, physically demanding, desert environment, where they were crowded into warehouses, storage buildings, and tents with little personal privacy and few amenities. No one knew that coalition forces eventually would win a quick war with relatively few battle casualties. Consequently, most troops did not fight a "four day war" but spent months isolated in the desert, under constant stress, concerned about their survival and their family's well-being at home, and uncertain about when they would return home.[2] Since the end of the war, readjustment disorders and posttraumatic stress disorder (PTSD) have been frequently reported among Persian Gulf veterans.[3,4,5]

Although exposure to chemical warfare (CW) and biological warfare (BW) agents has been hypothesized as a possible cause of ill health among the returning veterans, both a DoD Defense Science Board Task Force and the Institute of Medicine have concluded that there is no persuasive evidence that Iraq used CW/BW weapons or that there was exposure of U.S. troops.[6,7]

To provide protection against the lethal effects of CW nerve agents, troops were issued twenty-one 30 mg tablets of pyridostigmine bromide.[8] Pyridostigmine bromide has been

suggested as a cause of chronic illness in Gulf veterans. However, this Food and Drug Administration (FDA)-approved drug has been used since the 1950s in anesthesia and as a treatment for myasthenia gravis with no known long-term health effects. In addition, studies of this drug in low doses have not revealed any serious lasting side-effects.[9,10,11] Nonetheless, studies to evaluate the potential health effects of pyridostigmine, both alone and in combination with other agents, are ongoing.

Vaccines which protect against anthrax and botulism also have been mentioned as possible causes of ill health. Anthrax vaccine is a FDA licensed product. Although botulinum toxoid is not available as a licensed product, FDA approved its use by DoD as an Investigational New Drug after review of available safety information. Anthrax vaccine and botulinum toxoid have been given to military and civilian personnel worldwide for several decades without any long-term adverse effects.[7,12,13] Approximately 150,000 service members received anthrax vaccinations, while botulinum toxoid was administered to about 8,000 troops.

The surveillance and impact of infectious diseases during the Persian Gulf War have been summarized recently.[14] The major reported causes of acute morbidity were generally mild cases of acute diarrhea and upper respiratory disease. There was a decided absence of expected arboviral infections, particularly sandfly fever. Infectious diseases were not a major cause of lost manpower during ODS/S.

Since the Gulf War, thirty-one cases of leishmaniasis have been diagnosed among U.S. troops consisting of nineteen cases of cutaneous and twelve cases of viscerotropic leishmaniasis.[1] The nineteen cases of cutaneous leishmaniasis exhibited characteristic skin lesions. All but one of

the individuals with documented viscerotropic leishmaniasis have had characteristic, objective signs of disease, including fever, swollen lymph glands, and enlarged liver or spleen.[15]

Some Desert Storm troops may have been exposed to several potentially harmful environmental hazards, most notably smoke from 605 burning oil wells. The U.S. Army conducted an extensive health risk assessment (HRA) of smoke exposure which included methodology developed by the Environmental Protection Agency. The HRA determined long-term health risks to be minimal in part because of the nearly complete combustion of most chemical substances and the lofting of the smoke above ground level.[16,17]

Other potential environmental hazards that some service members may have been exposed to include: depleted uranium munitions, microwaves, chemical-agent-resistant-coating (CARC) paint vapors, various petroleum products, pesticides, and airborne allergens and irritants.[18] None of these exposures has been identified as a major cause of illness among Persian Gulf veterans, either because exposures involved small numbers of troops or because the agents are not known to cause the chronic symptoms reported by returning veterans.[6,7,14,16]

The Comprehensive Clinical Evaluation Program Process

Because of concern for the medical problems of Persian Gulf veterans and to better understand the nature of the diverse symptoms being reported, DoD established the CCEP on June 7, 1994. The CCEP provides a systematic, in-depth, medical evaluation for all military health care beneficiaries who are experiencing illnesses which they believe may be related to Persian Gulf deployment. Spouses and children of Gulf War veterans may participate in the CCEP if they are eligible for DoD health care.

Participants enroll in the program either by contacting their local military medical treatment facility or by calling a toll free number (1-800-796-9699) which provides information to individuals requesting medical evaluations. Every military medical treatment facility (MTF) has a designated CCEP physician coordinator who is a board-certified family practitioner or internal medicine specialist.

Developed by a multidisciplinary team of DoD and VA medical specialists, the CCEP provides a two-phase, comprehensive medical evaluation. Phase I is conducted at the local MTF and consists of a history and medical examination comparable in scope and thoroughness to an evaluation conducted for an in-patient hospital admission. The medical review includes questions about family history, health, occupation, unique exposures in the Gulf War, and a structured review of symptoms. Health care providers specifically inquire about the symptoms and exposures listed on the "CCEP Provider-Administered Patient Questionnaire." The medical examination focuses on patients' symptoms and health concerns, and includes standard laboratory tests (complete blood count, urinalysis, serum chemistries) and other tests as clinically indicated. Individuals who require additional evaluation after completing the MTF-level, Phase I evaluation and appropriate consultations may be referred to one of fifteen Regional Medical Centers (RMCs) for Phase II evaluations. Phase II evaluations consist of symptom-specific examinations, additional laboratory tests, and specialty consultations according to the prescribed protocol.

The DoD has established a Specialized Care Center (SCC) at Walter Reed Army Medical Center (Eastern Region), and has planned a second center for Wilford Hall Medical Center (Western Region) to provide additional evaluation, care and rehabilitation for CCEP

participants who are suffering from chronic, debilitating symptoms. An intensive 3 week evaluation and care program designed to restore participants to a maximum state of health and fitness is provided by the SCCs. A multidisciplinary team of physicians from various specialties, behavioral health psychologists, nurses, and physical and occupational therapists comprise the staff of the SCCs. The treatment program is modeled after multidisciplinary pain centers, which have proven effective in treating patients with chronic, debilitating syndromes.

Institute of Medicine

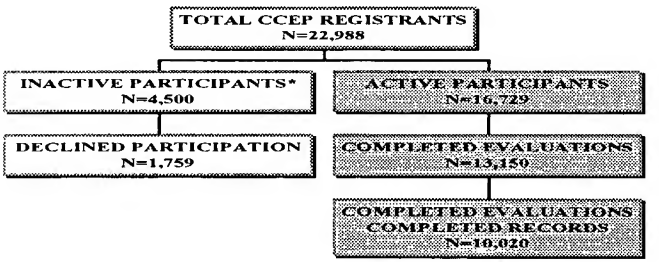
The DoD requested the Institute of Medicine (IOM) to serve as a consultant to review the CCEP. The IOM formed a panel of experts in epidemiology, occupational medicine, internal medicine, infectious diseases, psychiatry/psychology, community mental health, allergy/immunology, and other disciplines. The panel has met with the CCEP military physicians and other DoD representatives on two occasions to review both program process and results to date. The IOM initial report of December 1994 stated that the CCEP represented a thorough and systematic approach to the diagnosis of a wide spectrum of illnesses. The IOM recommended that a greater proportion of the CCEP evaluations be accomplished in Phase I to expedite the diagnostic process and facilitate continuity of care at the local level. A second IOM report is projected for the summer of 1995.

RESULTS

Program Status

Of the 16,729 participants who requested medical examinations through the CCEP, 10,020 records have been entered into the CCEP computerized database (Figure 1). MTFs send reports of finished medical evaluations to a central program management office where administrative staff and medical coders review records for completeness and accuracy of diagnostic coding before entering the data into a computerized database. Eighty-three percent (83%) of CCEP evaluations were completed at Phase I and seventeen percent (17%) at Phase II.

Figure 1. Disposition of CCEP Participants as of May 31, 1995



* Inactive Participants include those participants who wish to defer their medical evaluation until a later time.

Demographics

The demographic characteristics of 10,020 CCEP participants who have completed their evaluations are compared with the characteristics of all Gulf War veterans in Table 1.

Statistically significant differences ($p < 0.05$) are noted for each of the demographic categories with the exception of Hispanic ethnicity, rank, and Air Force affiliation. Additionally, CCEP participants are two years older on average than all Persian Gulf War veterans.

Table 1. Demographic Variables of CCEP Participants and Persian Gulf War Participants

Characteristics	CCEP Participants N=9,798[1]	Total PGW Participants N=697,000
Gender(%)		
Male	88	93
Female	12	7
Race(%)		
White	60	70
Black	30	23
Hispanic	6	5
Other	4	2
Rank(%)		
Enlisted	89	89
Officer	10	10
Other/No Data	1	1
Branch(%)		
Air Force	12	12
Army	78	50
Marine	4	15
Navy	5	23
Other/No Data	1	
Status(%)		
Active	82	83
Reserve/Guard	8	17
Other/No Data	10	
Age (Yrs)	34[2]	32[3]

[1] Includes only service members.
[2] The average age of the CCEP participants is as of June 1995.
[3] The average age of the PGW participants is as of June 1995.

Unit of Assignment

The approximately 700,000 personnel who deployed to the Persian Gulf War were assigned to military units designated by 13,448 different unit identification codes (UICs). The number of deployed personnel assigned to a single UIC varied from one person to several thousand (e.g., an aircraft carrier crew). Additionally, the Air Force used a limited number of large "administrative" UICs (for one example, one UIC had 20,978 personnel assigned). Some deployed personnel were subsequently assigned to multiple UICs throughout the theater.

Of the 10,020 CCEP participants with completed evaluations, 7,610 (76%) had UIC information available. These CCEP participants are representative of 2,725 different UICs, to which 443,898 service members (60% of the total force) were assigned. CCEP participants served in a very large number of different units, and eighty-five percent (85%) of UICs represented in the CCEP had four or fewer participants. Two hundred (200) individuals in the CCEP served in 62 different units (of 10 or more persons assigned) where CCEP participation rates were equal to or exceeded 10% of members of that UIC.

Self-Reported Exposures

The "CCEP Patient Questionnaire" asks the participants about exposures they experienced during the Persian Gulf War. This "self-reported' exposure information is dependent upon the participant's ability to recall events. Confirmation or validation of self-reported exposures was not possible using existing data sources for a given individual's exposures. Table 2 summarizes the most frequently self-reported exposures, including:

passive cigarette smoke (86%), diesel/other fuels (85%), pyridostigmine bromide tablets (70%), oil fire smoke (68%), and tent heater fumes (68%). Least often reported were suspected nerve gas/nerve agents (5%) and mustard/blistering agents (2%). The average number of positive exposure responses per CCEP participant was 11 of 20 potential exposures. Twenty-nine percent (29%) of the CCEP participants report they are current smokers, smoking an average of 16 cigarettes per day.

Table 2. CCEP Self-Reported Exposure History (n=10,020)

Exposures Reported By Participant	Positive Report	
	Number	%
Cigarette Smoke (Passive)	8,667	86
Diesel/Other Fuels	8,547	85
Pyridostigmine Bromide	7,020	70
Tent/Heater Fumes	6,784	68
Oil Fire Smoke	6,863	68
Personal Pesticide Use	6,400	64
Ate Non-U.S. Food	6,369	64
Had Anthrax Immunization	4,956	49
Solvent	4,508	45
Chemical Agent Resistant Coating (CARC) Paint	4,363	44
Other Paint	3,927	39
Microwaves	3,469	35
Bathed In/Drank Non-U.S. Water	3,199	32
Had Botulism Immunization	2,558	26
Taken Oral Medicine To Prevent Malaria	2,649	26
Ate Contaminated Food	2,050	20
Bathed In Contaminated Water	1,934	19
Depleted Uranium	1,415	14
Nerve Gas/Nerve Agents	501	5
Mustard Gas/Blistering Agents	234	2

Symptoms

The CCEP medical evaluation documents participants' chief health complaints and any other health complaints they may be experiencing. Table 3 summarizes the frequency distribution of positive responses to the "Provider-Administered Symptom Questionnaire."

Table 3. Symptom Frequency for CCEP Participants (N=10,020)

Symptoms Reported By Participants	Chief Complaint	Any Complaint
Fatigue	11%	47%
Joint Pain	11%	47%
Headache	8%	39%
Rash/Dermatitis	7%	29%
Memory Loss	4%	33%
Abdominal Pain/Gastrointestinal	2%	16%
Back Pain	2%	2%
Diarrhea	2%	18%
Dyspnea	2%	16%
Sleep Disturbance	2%	32%
Chest Complaints	1%	1%
Cough	1%	1%
Depression	1%	23%
Muscle Pain	1%	22%
Sinus Problems	1%	1%
Allergies	0%	0%
Bleeding Gums	0%	8%
Difficulty Concentrating	0%	27%
Dizziness	0%	0%
Hair Loss	0%	11%
Insomnia	0%	0%
Nausea	0%	0%
Weight Loss	0%	7%
People With No Chief Complaint	29%	
People With No Chief Or Any Complaint	11%	

The most frequently reported chief complaints were: fatigue (11%), joint pain (11%), headache (8%) and memory loss (4%). Among the reported symptoms, whether a chief or associated complaint, the most common symptoms from the symptom questionnaire included: fatigue (47%), joint pain (47%), headache (39%), memory loss (33%), sleep disturbance (32%), and difficulty concentrating (27%). The average number of reported symptoms for CCEP participants was five.

Diagnostic Categories

The distribution of CCEP diagnoses according to International Classification of Diseases-Ninth Revision, Clinical Modification (ICD-9-CM)[19] coding categories is shown in Table 4. The ICD-9-CM coding system is the standard method used in medicine for classification of diseases, injuries, and symptoms.

In the CCEP, "Psychological Conditions" (19%), "Symptoms, Signs and Ill-Defined Conditions" (17%), and "Musculoskeletal System" (17%) represent the most frequent diagnostic categories, accounting for 53% of all primary diagnoses. Additionally, eleven percent (11%) of participants had diagnoses involving "V Codes". "V Codes" are used to describe three groups of individuals in the CCEP: 1) those individuals without symptoms who request a medical evaluation, 2) those individuals with a normal medical evaluation, and 3) those individuals with a history of a preexisting condition but without a current illness. The average number of diagnoses per patient was three.

Of the 19% of CCEP participants with a primary diagnosis consisting of a "Psychological Condition," four diagnoses represent 59% of this category: tension headache, major and minor depressive disorders, and prolonged posttraumatic stress disorder.

Of the 17% of the participants with a primary diagnosis within the ICD-9-CM category of "Symptoms, Signs, and Ill-Defined Conditions," three diagnoses represent 63% of the total category and include: malaise and fatigue, sleep disturbance, and headache.

Table 4. Frequency Distribution of Primary and Any Diagnosis Among 10,020 Completed CCEP Evaluations (By ICD-9-CM Category)

Categories	Primary Diagnosis %	Any Diagnosis %
Psychological Conditions	19	37
Signs, Symptoms, and Ill Defined conditions[1]	17	41
Musculoskeletal	17	45
Healthy[2]	11	19
Respiratory System	7	18
Nervous System	6	18
Digestive System	6	22
Skin & Subcutaneous	6	20
Infectious Disease	3	9
Endocrine	2	11
Circulatory System	2	8
Neoplasm	1	3
Genitourinary System	1	6
Injury and Poisoning	1	3
Congenital Anomalies and Conditions of the Perinatal Period	<1	<1
Total	**100**	**N/A**

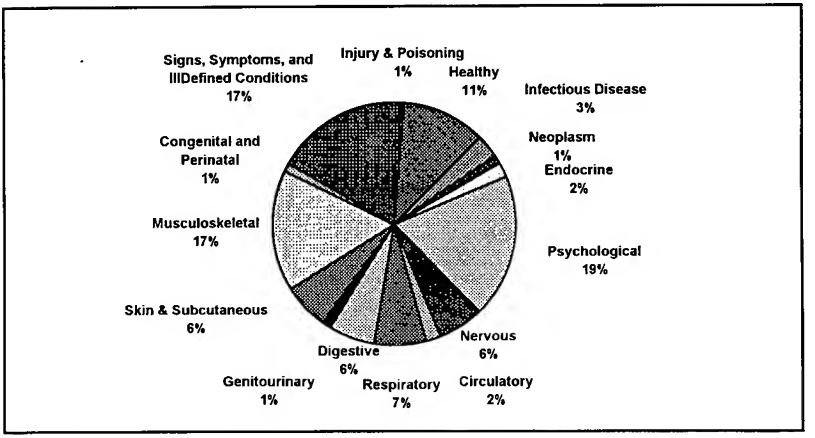
[1] Includes conditions categorized according to ICD-9 nomenclature consisting of cases for which no diagnosis is classifiable elsewhere; no more specific diagnosis can be made; signs or symptoms that prove to be transient; and, cases in which a more precise diagnosis was not available for any other reason.
[2] Includes "V Codes" which refer to CCEP participants who: a) are seeking consultation without complaint or illness, b) are not currently sick, and/or c) have a circumstance or problem which influences a person's health status but is not in itself a current illness or injury

Of the 17% of CCEP patients with a primary diagnosis of "Musculoskeletal and Connective Tissue Conditions," three diagnoses represent 51% of the category: pain in joint (s), osteoarthritis, and backache/lumbago.

Neoplasms represent 1% of all primary diagnoses. Malignant disease was diagnosed in 56 (0.6 %) of all CCEP participants. The most frequently diagnosed malignant neoplasms were skin cancers (15 participants) and lymphoma (12 participants).

The frequency distribution of primary diagnoses is shown in Figure 2.

Figure 2. Distribution of Primary Diagnoses Among 10,020 CCEP Participants.



The CCEP database includes the records of 136 dependent spouses of Persian Gulf War veterans and 81 children. The distributions of diagnoses among spouses and children are shown in Table 5 and Table 6.

Table 5. Frequency Distribution of Primary and Any Diagnosis Among 136 Completed CCEP Evaluations of Spouses (By ICD-9-CM Category)

Diagnoses	Primary Diagnoses %	Any Diagnoses %
Psychological Conditions	24	41
Signs, Symptoms, and Ill defined conditions[1]	11	35
Skin & Subcut. Tissue	10	24
Healthy[2]	10	20
Musculoskeletal/ Connective	9	32
Respiratory System	8	19
Nervous System	7	17
Digestive System	6	23
Endocrine	5	15
Genitourinary System	4	16
Infectious Diseases	2	7
Circulatory System	2	10
Neoplasm	1	4
Congenital Anomalies and Perinatal Conditions	1	3
Injury and Poisoning	0	0
Total	**100**	**N/A**

[1] Includes conditions categorized according to ICD-9-CM nomenclature consisting of cases for which no diagnosis is classifiable elsewhere; no more specific diagnosis can be made; signs or symptoms that prove to be transient; and, cases in which a more precise diagnosis was not available for any other reason.

[2] Includes "V Codes" which refer to CCEP participants who: a) are seeking consultation without complaint or illness, b) are not currently sick, and/or c) have a circumstance or problem which influences a person's health status but is not in itself a current illness or injury.

Table 6. Frequency Distribution Of Primary Diagnoses Among 81 Children of Persian Gulf War Veterans in the CCEP.

Diagnosis	Number
Healthy (Normal Exam)	17
Congenital Abnormalities*	15
Dermatitis, Eczema, Folliculitis, Acne	6
Asthma, Reactive Airway Disease	5
Other	5
Psychosis, Depression, Obsessive/Compulsive Disorder	4
Developmental Delay	4
Otitis Media	3
Upper Respiratory Infections	3
Rash	3
Nephritis, Vesicoureteral Reflux, Hydrocele	3
Tinea Captitis	2
Dermoid Cysts, Hemangiomas	2
Attention Deficit/Hyperactivity	1
Choroid Plexus Carcinoma	1
Anemia	1
Chronic Pneumonia	1
Chronic Diarrhea	1
Milk Allergy	1
Seizures	1
Insomnia	1
Static Encephalopathy of Childhood	1
Total	**81**

*Specific diagnoses include: Hydrocephalus (1), Glaucoma(1), Microsomia(1), Major Cardiac Anomalies(2), Cleft Palate(3), Trisomy 21(1), Fragile X Syndrome(1), Marcus-Gunn Syndrome(1), Pectus Excavatum(2), Left Hand Aphalangia(1), Omphalocoele(1) These congenital abnormalities are based only on children whose parents chose to enroll them in the CCEP. Because of the self-selected nature of the CCEP and the absence of information concerning all births of Persian Gulf veterans, this data can not be used to determine a rate of birth defects that can be compared to a non-Persian Gulf population.

Self-Reported Work Days Lost Due to Illness

The CCEP questionnaire asks how many days of work the participant has lost because of illness within the last 90 days. Over 80% of participants reported not missing any work days in the 90 days prior to the evaluation. The percentage of participants reporting "0 days lost" did not differ greatly between ICD-9-CM categories (range: 75-90%). Among

diagnostic categories, the average number of work days lost ranged from 1-8, with "Neoplasms" representing the disease category with the greatest number of missed work days.

Satisfaction with CCEP

Approximately 64% of CCEP participants (6429/10020) responded to the question at the conclusion of their medical evaluation: "Were you satisfied with the care you received in the program?" Ninety one percent (91%) or (5853/6429) replied affirmatively. The satisfaction rate among respondents who completed Phase II evaluations was 97% (695/720) compared to 87% (5581/6380) for those who completed their evaluations at Phase I.

DISCUSSION

Epidemiological Considerations

The CCEP represents a large case series of over 10,000 comprehensive, systematic health evaluations. However, several methodological limitations associated with the CCEP need to be understood to interpret findings appropriately in this population. Since the CCEP represents individuals who have self-selected to enter the program and excludes individuals ineligible for care through the military medical system, it may not be totally representative of the overall population of veterans with health concerns of PGW veterans as a group.

The CCEP has conducted an aggressive campaign to provide medical examinations to Persian Gulf War veterans who believe they are experiencing medical problems related to their participation in the Gulf War. This pro-active "case finding" effort has resulted in the systematic evaluation of 10,020 patients, to date, including approximately 1700 intensive

evaluations conducted at one of 15 tertiary care medical centers within the Military Health Services System. A case series, such as the CCEP, is not definitive in determining risk factors, causality or specifically defining associations, particularly when self-reported exposures cannot be validated. However, the CCEP does have utility in detecting a potential new clinical syndrome and in describing the nature of symptoms and illnesses in a very large group of veterans.

Comparison Group Selection

From an epidemiological perspective, either non-deployed Gulf War-era veterans or Gulf War-era veterans who experienced some other deployment represent appropriate groups for comparative purposes. Studies of outpatient diagnoses for a population of non-deployed, Persian Gulf War-era veterans, while in progress, are incomplete at this time. However, for the purposes of the CCEP, until more definitive comparisons are made, use of both population-based surveys and examinations (e.g., National Ambulatory Medical Care Survey, National Institute of Mental Health Epidemiologic Catchment Area Studies, etc.), and other studies of symptoms and diagnoses in ambulatory patients, provide useful comparative information. Formal research efforts (which include appropriate control or comparison groups in their study design) by the Departments of Defense, Veterans Affairs and Health and Human Services (HHS) will, together with the CCEP, further characterize the health status of PGW veterans.

Demographics

Demographic variables of CCEP participants were compared with all who deployed to Operation Desert Shield/Desert Storm. A statistically significant difference was noted for each of the demographic variables with the exception of rank, Hispanic ethnicity, and Air Force affiliation. Given the self-selected nature of participants in the CCEP and eligibility criteria for access to DoD health care facilities, it is difficult to draw any meaningful conclusions from these differences, other than to say that CCEP participants are a non-random sample of the Persian Gulf War veteran population. The CCEP does, however, represent a somewhat balanced cross-section of all who deployed to the Gulf and there appear to be no unique characteristics among CCEP participants. Well-designed epidemiologic studies that compare the CCEP sub-population with an appropriately matched control population will provide the best information on which to base conclusions regarding demographics.

Unit Identification Codes - Specific Participation Rates

UIC specific CCEP participation rates indicated a low rate of CCEP participation in the great majority of UICs (mean 1.7 per 100 service members). This low rate of participation was present across a large range of UICs involving all services. The low "UIC-specific CCEP participation rates" across the wide range of UICs suggest that geographic clustering of illness did not occur. Although there appears to be no unique clustering of CCEP participants by UICs, the wartime and post-war experience of personnel within UICs with relatively higher rates of CCEP participation warrant further investigation.

Use of Unit Identification Codes to Validate Self-Reported Exposures

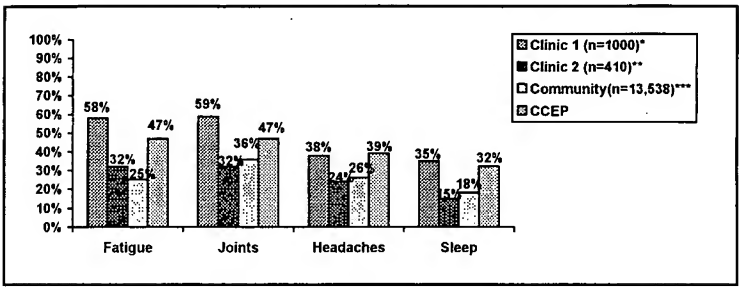
Several of the potential exposures which occurred in the Persian Gulf (as self-reported by CCEP participants) were confined to units in specialized occupations or certain geographic locations. For example, malaria prophylaxis was provided only to selected units based upon geographic location. Similarly, anthrax and botulinum immunizations were restricted to certain units which were deployed forward in ODS/S. Analysis of the CCEP population by UIC-specific locations and military occupational specialty groups may enable validation of these and other exposures. The U.S. Army Center for Health Promotion and Preventive Medicine (USACHPPM) is currently integrating exposure data sets, troop movement data, and satellite imagery of the oil well fire period into a Geographic Information System (GIS) model thereby enabling spatial analyses. Additionally, analysis of classified and declassified operational, intelligence, medical sources of information, research databases, as well as anecdotal accounts of veterans will be correlated with findings of the CCEP and GIS to determine exposure relationships throughout the Persian Gulf theater of operations.

Symptoms

As shown in Figure 3, four of the most common symptoms reported by participants in the CCEP are also prevalent in the U.S. population as a whole and among patients in general medical practice. Three published surveys of outpatient practices are informative.[20,21,22]

Because the characteristics of the study groups and duration vary, exact comparisons of symptoms and illnesses can not be made among the three studies.

Figure 3. Common Symptom Prevalence as Reported in 3 Studies of Outpatient Practice in the United States, as Compared with CCEP



*Clinical Survey 1 =1000 patients presenting for care at four primary care clinics in the U.S.
**Clinical Survey 2 = 410 patients attending a military general medicine clinic
***Community Survey 3 = Random survey of 13,538 persons in four communities in the U.S.

There appears, however, to be strong consistency of reported symptoms between large population studies of outpatient medical clinics and symptoms reported by CCEP participants. Referring to Figure 3, fatigue was reported by 22-58% (CCEP 47%) of respondents; joint pains by 26-59% (CCEP 47%); headaches by 21-37% (CCEP 39%), and sleep complaints by 15-35% (CCEP 32%). Not shown in Figure 3, but also very common in these surveys, were dyspnea for 14-32% (CCEP 16%) and abdominal pains for 11-24% (CCEP 16%).

The similarity of these particular CCEP symptoms in the U.S. general clinic population is further confirmed by examining data from the National Ambulatory Medical Care Survey (NAMCS). This national sample of medical clinics in the United States reported, that in 1989, the estimated number of outpatient visits in the United States was: 7 million visits for fatigue;

9.6 million visits for headaches; 17 million visits for joint pains; 14 million visits for skin rash; and 7 million visits for depression.

Patients commonly report experiencing multiple symptoms. Studies have shown that when patients complete symptom checklists one third of patients complain of 0-1 symptoms, one-third complain of 2-3 symptoms, and one-third complain of 4 or more symptoms.[21,23] Research conducted by Kroenke et al indicates that typical outpatients will endorse a median of 4 symptoms as bothersome.[22,24] CCEP patients reported an average of 5 symptoms per patient.

"Symptom syndromes", i.e., illnesses manifested solely by combinations of symptoms with no consistent objective findings on physical examination or positive laboratory abnormalities and for which an adequate etiologic explanation is yet to be determined, are common in clinical practice and the general population. Symptom syndromes include entities such as irritable bowel syndrome, fibromyalgia, Chronic Fatigue Syndrome (CFS) and depression. Moreover, the overlap of specific symptoms can be considerable.[25,26,27,28,29] For example, Table 7 compares the frequency of various symptoms seen in CCEP patients and three other common symptom syndromes.

Table 7. Prevalence of Various Symptoms in 3 Common Symptom Syndromes Compared with CCEP Patients

Symptom %	CCEP Patients (%)	CFS(%)	Fibromyalgia(%)	Major Depression(%)
Fatigue	47	100	85+	80+
Myalgia	47	40-50	100	40+
Headache	39	35-85	44-88	70+
Memory	33	50-85	-	80+
Sleep	32	15-90	56-80	80+
Depressed Mood	23	46-85	20-71	90+

Physical symptoms in both clinic patients and the general population frequently lack a clear-cut or definitive physical explanation or "cause." Four community-based studies have shown that 20 to 75% of symptoms lack an association with a definitive diagnosis after a medical evaluation.[20,22,23,24] The best estimate is that about one-third of symptoms cannot be linked to a defined diagnosis.

A recent study by the Centers for Disease Control and Prevention (CDC) compared the prevalence of symptoms in Persian Gulf veterans to non-deployed, Persian Gulf-era veterans.[30] Preliminary findings indicated that chronic symptoms, similar to those seen in CCEP participants, were reported more commonly by Persian Gulf veterans than non-deployed, Persian Gulf-era veterans. Comprehensive medical evaluations by CDC physicians and a review of medical records for fifty-nine Persian Gulf War veterans in the initial case series did not identify any consistent physical or laboratory abnormalities. A case-control study is currently being completed to compare symptoms and illnesses in deployed and non-deployed Persian Gulf War service members.

Existence of a Unique Illness

DoD physicians have diagnosed a wide range of medical conditions commonly seen in general medical practice, but have found no clinical evidence for a unique illness among CCEP participants. The large number of patients participating in the CCEP, the thoroughness of the evaluations, and the clinical impressions of CCEP physicians are the primary basis for forming conclusions regarding the existence of a new or unique condition or syndrome. Although

CCEP physicians have not found clinical evidence for a unique illness or syndrome in examining individual patients, analysis of demographic, exposure, symptom and diagnostic results is useful in characterizing the nature of illnesses being experienced by Persian Gulf veterans participating in the CCEP.

CCEP Diagnoses Relative to Other Ambulatory Care Studies

The National Ambulatory Medical Care Survey (NAMCS) is considered the most representative study of outpatient medical practice in the U.S.[31] It must be noted, however, that the NAMCS includes not only individuals with persistent symptoms but also patients with acute illnesses or injuries, healthy persons needing school physicals, employment or insurance examinations and other types of "walk-in" clinic visits. In contrast, the CCEP is more likely to include individuals with persistent symptoms and, as such, does not include patients whose symptoms typically are short-lived. Additionally, differences in the proportion of male and female participants in the two populations would contribute to differences in the size and composition of diagnostic categories. Therefore, because the populations differ, even general comparisons should be made with caution.

Generally speaking, the frequency distribution for a number of the ICD-9-CM codes appear similar in the NAMCS and CCEP patient populations (see Table 8), except for "Psychological Conditions," "Respiratory System," "Genitourinary System,"

"Musculoskeletal System," "Symptoms, Signs, and Ill-Defined Conditions," "Injury and Poisoning," and"V Codes" (Healthy).

Table 8. Frequency Distribution of Principal Diagnoses in U.S. Ambulatory Care Survey* (NAMCS) and in the CCEP

Frequency Distribution Of Principal Diagnoses In United States Ambulatory Care (NAMCS) And In The CCEP			
Primary Diagnoses	**ICD-9-CM**	**NAMCS[31]**	**CCEP**
		--Percent Distribution--	
Infectious Diseases	001-139	3.5	2.9
Neoplasms	140-239	2.4	1.1
Endocrine	240-279	1.3	1.8
Psychological conditions	290-319	6.0	18.6
Nervous System	320-389	6.5	5.4
Circulatory System	390-459	3.1	2.2
Respiratory System	460-519	11.7	6.7
Digestive System	520-579	3.8	6.4
Genitourinary System	580-629	8.1	1.5
Skin & Subcut. Tissue	680-709	6.8	6.3
Musculoskeletal System & Connective Tissue	710-739	6.8	17.0
Symptoms, Signs & Ill-Defined Conditions	780-799	4.1	17.5
Injury And Poisoning	800-999	10.7	0.7
"V Codes" (Healthy)		19.9	10.9
All other Diagnoses[1]		2.3	1.0
Unknown or blank[2]		2.3	0.0

*NAMCS data for ages 25-44

[1]Includes diseases of the blood and blood-forming organs (280-288)/complications of pregnancy, childbirth, and the puerperium (630-676); congenital anomalies (740-759); and certain conditions originating in the perinatal period (760-779).

[2]Includes blank diagnoses, uncodable diagnoses, and illegible diagnoses.

For purposes of general comparison, respiratory system diagnoses appear to occur more commonly in the NAMCS (11.7% vs. 6.7%), probably because upper respiratory illnesses (URI's), such as the common cold, are one of the leading reasons for outpatient visits in the United States. Because URI's typically resolve in a week or less, very few patients with

URI's would be represented in the CCEP. Injury and poisoning account for 10.7% of diagnoses in the NAMC but only 0.7% in the CCEP. Such patients are acutely ill or injured, come in promptly for care, and are treated within a matter of hours to a few days. Thus, they would be common in an office practice seeing acute and chronic patients but would be rare in a sample of patients with persistent symptoms, such as the CCEP. "V Codes" (Healthy) appear to be more common in the NAMCS because the leading reason for outpatient visits in the United States is general medical examinations, such as routine physicals for school, employment, or insurance purposes or annual "checkups" for health maintenance reasons. Asymptomatic patients coming in for routine physicals or simple checkups would be less prevalent in the CCEP. The higher percentage of genitourinary diagnoses in NAMCS may reflect the larger proportion of women in that survey compared to the CCEP and an increased prevalence of gender related diagnoses, such as bladder infections.

On the other hand, three diagnoses - "Psychological Conditions," "Symptoms, Signs and Ill-Defined Conditions", and "Musculoskeletal System" are more common in the CCEP than in NAMCS (53% vs. 17%). Possible explanations for the apparently higher prevalence of these diagnostic categories in the CCEP warrant discussion, which follows.

Psychological Conditions

Psychiatric disorders such as depression, anxiety, and somatoform disorders are common in primary care, existing in 25-35% of all patients presenting for care in the outpatient setting.[32,33] Overall prevalence of psychological conditions among CCEP patients (19% primary, 37% any diagnosis) may be somewhat higher than that found for other groups

of health care seeking individuals (Table 9) in which structured psychiatric interviews were used.

Table 9. Prevalence Of Psychological Conditions In CCEP Participants Compared To Other Community And Primary Care Cohorts

Sample Source	Prevalence of Psychological Conditions (All Conditions)
Midtown Manhattan Study (Community)	23%*
NIMH Epidemiologic Catchment Area Studies (Community)	18-27%
Primary Care	25%-35%
CCEP	19-37%

*percentage of respondents with "serious impairment".

The most common psychological conditions among CCEP patients are: somatoform problems, especially tension headache; nonspecific, mild, or stress-related anxiety and/or depression; posttraumatic stress disorder, and alcohol-related disorders. None of these disorders was noticeably more prevalent than available figures from previous community and primary care based studies (see Table 10).[34,35]

Table 10. Most Common Psychological Conditions Among CCEP Participants, General Population And Primary Care Patients

Psychological Condition	CCEP		General Pop'n	Primary Care
	Primary Dx	All Dx		
Somatoform Problems	5%	15%	---	---
Somatization Disorder	0.8%	2%	0.1-1%	1-5%
Tension Headache	4%	12%	20%	---
Mood Depression	6%	12%	4-6%	15-25%
Major Depressive Disorder	2%	3%	2-5%	5-14%
Mild Depressive Syndrome	4%	8%	7%	---
Anxiety Disorders	4%	8%	7%	5-15%
Posttraumatic Stress Disorder	3%	5%	1-14%	---
Mild Anxiety Syndromes	0.6%	2%	---	---
Adjustment Disorders	2%	4%	---	---
Substance Related Disorders	0.5%	2%	---	---
Alcohol Related Disorders	0.4%	2%	4-6%	6%

Psychological conditions may be more common in the CCEP because patients with persistent or unexplained symptoms have high rates (50% or more) of underlying mood or anxiety disorders. This need not always mean that the symptoms are caused by the mood or anxiety disorder since it is possible that depression or anxiety can be a consequence of persistent, disabling physical symptoms. Nonetheless, the mood or anxiety disorders that coexist in half or more of such patients can further aggravate such symptoms through worsening sleep, increased fatigue, lowered pain tolerance, and mental suffering.

Ill-Defined Signs and Symptoms

Approximately 17% of CCEP participants have primary diagnoses categorized by the ICD-9-CM as "Signs, Symptoms and Ill-Defined Conditions." Although an illness or symptom may fall in the 780-799 ICD-9-CM code range, that code may represent a well-defined condition not classified elsewhere (e.g., obstructive sleep apnea) or a nonspecific laboratory abnormality (e.g., elevated sediment rate). Also, patients with persistent symptoms in whom physical examination and diagnostic testing is normal often end up with a "symptomatic" diagnosis (e.g., lower back pain, headache, sleep disturbance, fatigue) rather than a more precise, anatomic or pathophysiologic diagnosis.

Depression and anxiety show a particularly strong association with unexplained or ill-defined physical symptoms. Studies have demonstrated that ill-defined (compared with better-defined) symptoms or syndromes tend to occur much more frequently in individuals with common, treatable anxiety and depressive disorders.[21,22] Treatment of depression and anxiety is known to decrease the severity of physical symptoms.

The higher proportion of "ill-defined diagnoses" (as compared with the NAMCS) is consistent with the earlier observations that the CCEP preferentially selects for patients with persistent symptoms and underrepresents those with acute, self-limited illnesses.

Musculoskeletal Conditions

Seventeen percent (17%) of CCEP participants had a primary diagnosis of musculoskeletal/connective tissue disorders. Forty-two percent (42%) of the diagnoses in this category consist of pain in joint, osteoarthritis, and unspecified arthropathy. Osteoarthritis is commonly the chronic result of occupational and recreational overuse injuries. Such injuries frequently occur as a consequence of the physical activity associated with military operations and training. Review of military disability evaluation system information confirms that musculoskeletal conditions are a leading category of disability in the Armed Forces. Musculoskeletal impairments are among the most common and disabling of medical disorders.[36] Therefore, the increased prevalence of musculoskeletal conditions in the CCEP relative to NAMCS may be related to the physical demands military service.

Additional Diagnostic Considerations

Infectious diseases and conditions associated with symptoms of fatigue, sleep disturbances and memory loss are of special concern and warrant further discussion.

Infectious Diseases

The threat to deployed military personnel posed by infectious diseases was recognized and preparations were made from the earliest stages of Operation Desert Shield.[37] Specific

infectious diseases observed in U.S. troops during Operations Desert Shield/Storm conformed with expected disease threats. Data suggest that overall exposure to recognized pathogens was quite low. Furthermore, it suggests that no route of infection, other than ingestion of locally-procured food, was common. The reported incidence of infectious diseases observed during the Operations is relevant to evaluation of current health complaints of Gulf War veterans.

Leishmaniasis has been one of the infectious diseases of particular concern in evaluating Persian Gulf veterans. Since the Gulf War a total of 31 cases of leishmaniasis have been identified among Persian Gulf veterans (12 cases of viscerotropic disease and 19 cases of cutaneous disease).[38] Virtually all of these cases were identified prior to initiation of the CCEP, and none was identified by the CCEP. All but one of the cases were characterized by the presence of objective findings on physical examination or screening laboratory assays. The low incidence of leishmaniasis during and immediately after Operations Desert Shield/Storm, the absence of other sandfly-borne diseases in our troops, and the low prevalence of objective findings pointing to leishmanial disease among 10,000 CCEP patients, all indicate that viscerotropic leishmaniasis plays no significant role in the current complaints of Gulf War veterans.

The CCEP itself has identified a wide variety of infectious diagnoses. Of these, by far the largest group has been fungal infections of the skin due to fungi common in the United States. Virtually all of the remaining infections have represented common illnesses, such as sinusitis, diarrheas, and a few cases of viral hepatitis, not specific to the Persian Gulf region.

The overwhelming majority of these diagnoses represent incidental diagnoses which would not explain persistent systemic complaints.

Fatigue

Chronic Fatigue Syndrome (CFS) has been suggested as a unifying diagnosis for unexplained illnesses among Persian Gulf veterans. Complaints of chronic fatigue lasting greater than six months are not uncommon in the general population seeking medical care [39,40] However, patients meeting the Chronic Fatigue Syndrome case definition are relatively rare. Records of 4.6% (464/10,020) of the CCEP patients carried a primary diagnosis of "fatigue" (ICD-9-CM 780.7 - 780.79). These records were reviewed to determine if they met the 1994 CDC case definition of CFS. Forty-two of the 464 participants with a primary diagnosis of fatigue (9 %) met the case definition. This represents a prevalence in the CCEP study population of 0.42% (42/10,020). This rate is similar to the prevalence rate of 0.3% reported in a general medical population.[40]

An additional syndrome Fibromyalgia Syndrome (FMS) is often mentioned together with CFS because fatigue is prominent among the somatic symptoms seen with this entity. The definition for FMS has been published by the American College of Rheumatology.[41] CCEP records were examined for FMS in both primary and secondary diagnoses. Fibromyalgia was found as the primary diagnosis in 78 of 10,020 individuals (0.78%) and in 175 individuals (1.75%) in primary or secondary diagnoses. These prevalence rates are consistent with those seen in the U.S. population.

Sleep

Sleep disturbances represent medical conditions which can be systematically evaluated and treated. Sleep disturbance was the fifth most common complaint among CCEP participants, with 32% reporting this symptom. Epidemiological surveys of the general population yield similar results of subjects reporting sleep problems ranging from 30-40%. A recent Gallop survey based on 1,950 phone interviews found that 36% of Americans suffer from some type of sleep problem.[42] Substantial numbers of sleep disorders (ICD-9-CM 307.4, 780.5) were found in the CCEP participants, with 357 (4%) having a sleep disorder as the primary diagnosis. Sleep disorders most frequently diagnosed in the CCEP participants were sleep apnea and disorders involving initiating and maintaining sleep (insomnias). The prevalence of sleep disorders in CCEP participants did not exceed that expected for the general population.[43,44]

The common sequelae resulting from these sleep disorders is chronic sleep deprivation. It is well documented that chronic sleep deprivation can lead to various physical and psychological complaints, many of which are the same as described by the CCEP participants (See Table 3).[45] Evidence from epidemiological studies suggests a strong association between sleep and other somatic complaints, though there is no clear cause-and-effect relationship.[46]

Memory Problems

Memory problems are a frequent complaint associated with the medical conditions diagnosed among the Persian Gulf veterans. They are the fourth most common symptom

among CCEP participants. Memory is not an isolated function, but rather a complex of neurobiological and neuropsychological processes. It represents one of the neurobehavioral functions most sensitive to central nervous system disruption.[47] In-depth neuropsychological evaluations in the CCEP population have identified no evidence of an increased prevalence of neurologic etiologies for memory loss. Organic mental disorders (ICD-9-CM code 310.1, 310.1, 310.8, 310.9) confirmed by neuropsychological testing and evaluation were found in only 0.6% of CCEP participants, which is less than expected within the general population.

Patients with depression, sleep disorders, chronic fatigue, and chronic pain often complain of memory problems.[48,49,50] These medical conditions represent potentially treatable causes of memory complaints.

Diagnoses in Spouses and Children

Some Gulf War veterans have expressed concern that the health of their spouses and children may have been affected by their military service in the Gulf War. The spectrum of diagnoses in spouses and children (Tables 5 and 6) spans a wide range of organ systems. Although relatively few spouses have enrolled in the CCEP, the distribution of their diagnoses is consistent with the types of conditions commonly seen in clinical practice and seen in CCEP participants overall. Likewise, the diagnoses of children enrolled in the CCEP appears to be similar to that seen in pediatric practice. As the number of these self-referred spouses and children in the CCEP cohort is small, comparisons with other study groups would not yield worthwhile results.

Reproductive Health Concerns

Some Persian Gulf War veterans have reported adverse birth outcomes and reproductive problems. The CCEP includes 15 children with congenital abnormalities whose parents chose to enroll them in the program. These birth defects span a wide range of conditions which are not concentrated in any single organ system or congenital syndrome. Because of the self-selected nature of the CCEP and the absence of information about a representative sample of births of all Persian Gulf veterans, data are insufficient to determine a rate of birth defects that can be compared to a non-Persian Gulf population.

Investigations by state and national public health agencies and DoD have identified no elevated rates or unusual patterns of birth defects in babies born to Gulf War veterans or their spouses.[51,52,53] In response to veterans' concerns, specific questions regarding reproductive history were added to the revised CCEP questionnaire in February, 1995. Less than 8% of the CCEP records in this report contained self-reported reproductive history; therefore, analysis has been deferred to future reports. However, to date, the birth defects which have been documented in pediatric CCEP participants are few in number and dissimilar in type. Population-based reproductive health outcome studies currently in progress will provide a more definitive assessment of possible reproductive health consequences related to service in the Persian Gulf War.

Disability

As an approximation of severity of morbidity and/or acute disability, "lost work days due to illness in the past 90 days" were obtained. Most CCEP participants (81%) did not

report missed work due to illness or injury during the 90 days prior to their initial evaluation. The distribution of "lost work days" did not vary substantially among different disease categories. Seven percent of CCEP participants self-reported missing more than one week of work due to illness during the previous 90 days. Sixty-one participants reported not working during the entire 90 day period; 19 individuals had psychological conditions and 12 individuals had diagnoses in the "Symptoms, Signs and Ill-Defined Conditions" category. Although absenteeism is only one marker for the assessment of disability, the data suggest that few CCEP participants are experiencing disabling symptoms severe enough to interfere with work.

Patient Satisfaction

In an effort to assess satisfaction with the CCEP, participants were asked whether or not they were satisfied with the CCEP program. Of the 65% of patients who answered the question regarding satisfaction, 91% were satisfied with the care received in the program. Percentages did not differ among the subsets of patients with a 780-799 ICD-9-CM diagnosis (90%), and "V Codes", (healthy) diagnosis (92%), or a mental disorder diagnosis (90%). By comparison, in the largest national study of outpatient satisfaction to date (the Medical Outcomes Study involving 17,671 patients), the percentage of patients rating their care as very good to excellent was 87% overall, but in managed care settings (the system most similar to the military health care system), the percent satisfied was somewhat lower.[54] In general, the satisfaction ratings concerning the care received in the CCEP are similar to satisfaction surveys in the civilian sector.

Additional Research Efforts

The CCEP and the VA Persian Gulf Health Registry are providing information about the types of symptoms and illnesses experienced by Gulf War veterans. However these clinical programs are not able to fully determine the prevalence, incidence, or risk factors of disease related to ODS/S deployment. Therefore, an extensive research program has been initiated by DoD, DVA, and HHS which will complement registry findings.[55] Among the efforts in process, are three major epidemiological studies being conducted by the Naval Health Research Center, the CDC, and the DVA. The Naval Health Research Center, San Diego, CA (in collaboration with the DVA, HHS, and the University of California), is conducting a series of epidemiological studies of active duty military personnel. Studies include personal interviews and physiologic testing of 750 ODS/S veterans and 1500 non-deployed Gulf-era veterans; analysis of the hospitalization records of 1.2 million service members; and review of pregnancy outcomes among Gulf War veterans and their spouses. Initial findings from these studies are expected in late 1995.

The DVA Environmental Epidemiology Service, Washington, DC, (in collaboration with DoD and HHS) is planning a random survey of 15,000 veterans who served in the Persian Gulf and 15,000 "control" era veterans. This mail/telephone survey is designed to: a) describe symptomatology experienced after Gulf service; b) assess the current health status of veterans and their family members, including reproductive health; and, c) evaluate the risk of potential environmental exposures.

The CDC is also planning to investigate the prevalence of reported symptomatolgy, illnesses, and exposures among Persian Gulf service members who list Iowa as their home of record.

Other ongoing research studies will further assess reproductive health, evaluate new diagnostic tests for infection, and study the health effects of exposure to depleted uranium and possible interactive effects of chemical exposures. This extensive research program will provide a comprehensive evaluation of the health consequences of Persian Gulf service and will contribute to the development of programs to protect the health of military personnel during future deployments.

The information maintained in the CCEP database constitutes a large case series, and was not designed to be a research study. Nevertheless, the CCEP database provides valuable descriptive information and, as such, is useful for generating hypotheses for future research. Once privacy act provisions have been met which ensure the protection of individual participants, the entire CCEP data set will be placed in a format that will allow access to a broad range of scientific investigators. The DoD anticipates working on this project with the National Technical Information Service through the Defense Technical Information Center.

Individual and Group Response to Environmental Hazards as a Factor Contributing to Health Consequences Among CCEP Participants

Fatigue, stress, fear, sleep disturbances, posttraumatic stress symptoms, anxiety and depression are common conditions which have been observed among military populations after participation in armed conflicts [56,57 58 59 60] as well as persons who have experienced

natural and manmade disasters.[61] Such physical and psychological effects may persist long after a disaster has occurred. War is one of the most complex of the man made environmental disasters.[62] Persian Gulf War veterans experienced the hazards of war which are always associated with combat. In addition, they experienced the unique environmental exposures and threats of exposure of the Persian Gulf, (e.g., the Kuwaiti oil well fires). Combat stressors (environmental exposures and the threat of environmental hazards) are complex events with multiple physiological, psychological and social responses in individuals who experience them.[63]

Service in the Persian Gulf involved numerous stressors, i.e., infectious diseases, chemicals, radiation, smoke from oil well fires, and possible reactions to prophylactic drugs and vaccines. For many, the threat of an environmental hazard was ever present.

It is clear that the Persian Gulf War experience itself, combined with an atmosphere of uncertainty concerning possible health consequences, has resulted in stress for many CCEP participants. The experience of environmental threat or the threat of a disaster can be very important in determining chronic stress and mental health effects. People may experience symptoms which are a direct result of exposure and/or threat of exposure. An individual will often link symptoms to an exposure threat.

The CCEP has identified/confirmed symptoms and diagnosable diseases, both physical and psychological, that would be expected in a general population. Psychiatric diagnoses account for 19% of all primary diagnoses in the CCEP. These diagnoses reflect both expected rates in the general population and the influence of chronic stress on this group of patients as a result of their concerns about exposures. Further research on the relative impact of the Gulf

War experience in terms of development of medical conditions seen in the CCEP will require epidemiologic studies involving appropriate comparison groups.

CONCLUSIONS

The large size of the CCEP cohort and the thoroughness of CCEP examinations provide considerable clinical insight for understanding the nature of illnesses and health complaints being experienced by this group of veterans. However, self-selection of patients, differential eligibility, recall bias, inability to validate self-reported exposures, and lack of an appropriate control group limit the generalization of these findings to other Gulf War veterans.

The CCEP has conducted an aggressive campaign to provide medical examinations to Persian Gulf War veterans who believe they are experiencing medical problems related to their participation in the Gulf War. This pro-active "case finding" effort has resulted in the systematic evaluation of 10,020 patients, to date, including approximately 1700 intensive evaluations at one of 15 tertiary care medical centers within the Military Health Services System. The large number of patients participating in the CCEP, the thoroughness of the evaluations, and the clinical impressions of CCEP physicians are the primary basis for conclusions regarding the lack of existence of a new or unique condition or syndrome.

Based on the CCEP experience to date, there exists no clinical evidence for a new or unique illness or syndrome among Persian Gulf veterans. DoD physicians have diagnosed a wide range of medical conditions commonly seen in general medical practice. The results of the CCEP are consistent with conclusions of a National Institutes of Health Technology Assessment Workshop that "no single disease or syndrome is apparent, but rather multiple

illnesses with overlapping symptoms and causes." Although CCEP physicians have found no clinical evidence for a unique illness or syndrome, the analysis of demographic information, exposure data, symptoms, and diagnostic results, are useful in characterizing the types of illnesses being experienced by Persian Gulf veterans participating in the CCEP.

In general, there appear to be no unique distinguishing characteristics of CCEP participants. CCEP participants served in a large number of units during the Persian Gulf. Preliminary analysis indicates no apparent clustering of CCEP participants on the basis of unit of assignment during the Gulf War. The exposures which CCEP participants describe span a wide range of occupational and environmental chemical/physical agents, vaccines and medications. Confirmation of these exposures was not within the scope of the CCEP, since the primary objective of the exposure questionnaire was to assist the physician in the diagnosis of the patient's medical condition. However, in specific instances, exposures are known to have been limited to relatively small numbers of individuals (e.g., depleted uranium, malaria prophylaxis, and botulinum toxoid).

CCEP participants commonly report experiencing symptoms of fatigue, joint pain, headache, and sleep disturbances. Review of studies of patients with similar chronic health complaints seeking primary care in the U.S. indicate these symptoms are routinely reported and are not unique to CCEP participants. Although the types of symptoms being experienced by CCEP participants are not unique, studies using appropriate control populations will determine whether these symptoms are associated with greater illness in subsets of Persian Gulf veterans than might be expected.

The CCEP has identified a wide range of primary diagnoses commonly seen in clinical practice (e.g., tension headache, migraine headache, fatigue, osteoarthritis, back pain, depression and stress related conditions). Using standard ICD-9-CM coding criteria, 51% of the CCEP diagnoses can be classified as "Psychological Conditions," "Signs, Symptoms, Ill-Defined Conditions," and "Musculoskeletal & Connective Tissue." Review of NAMCS data suggests that these diagnostic categories may be over represented in the CCEP. Potential explanations for these differences include, but are not limited to: 1) aggressive "case finding" which has attracted Persian Gulf war veterans with chronic, non-specific symptoms; 2) selection of individuals with background physical conditions (musculoskeletal injuries) associated with the physical demands of military service; 3) use of a structured, in-depth protocol to diagnose physiologic and psychological conditions which might otherwise not be evident in the course of routine, primary care; and, 4) factors directly related to the Persian Gulf War experience, such as exposure to stressful circumstances.

Concern regarding the possible existence of "unexplained illnesses" was a major consideration in the design of the CCEP. Although CCEP physicians have not identified a unique illness or syndrome, 17% of CCEP primary diagnoses can be categorized as "Signs, Symptoms and Ill-Defined Conditions" according to ICD-9-CM coding criteria. It should be noted that these diagnoses refer to a variety of conditions (well-defined conditions not classified elsewhere in the ICD-9-CM system, generalized symptoms, nonspecific findings, and abnormal laboratory tests) commonly encountered in primary care medical practice. As previously discussed, physical symptoms in both clinic patients and the general population frequently lack a clear-cut or discrete physical explanation or "cause." Coding of a diagnosis

within the category of "Signs, Symptoms and Ill-Defined Conditions" primarily reflects limitations in diagnostic and/or coding criteria rather than an impression as to whether or not the condition can be explained.

Severe disability measured in terms of lost work days is not a major characteristic of CCEP participants. Some CCEP patients with severe disability may benefit from participation in special programs which focus on rehabilitation, restoration of function and promotion of general well being. The DoD has established Specialized Care Centers, staffed by interdisciplinary teams, to provide such programs.

The broad issue of exposure to combat stressors (environmental exposures and threat of environmental hazards) is complex in terms of psychological, physiological and social responses of those who experience them. Research to assess the relative impact of the Persian Gulf experience as a contributing factor in the development of medical conditions seen in the CCEP will require epidemiologic studies involving appropriate comparison groups.

The CCEP has documented symptoms and confirmed diagnoses in over 10,000 individuals. Results of questionnaires and surveys suggest that CCEP participants have generally been very satisfied with the care they have received. DoD will continue to provide comprehensive, quality health care to eligible Persian Gulf veterans and will maintain an ongoing search for unique symptom/illness patterns. The Department is committed to an ongoing exchange of health information with other government agencies and Persian Gulf veterans to further understand this important public health issue.

[1] Persian Gulf Veterans Coordinating Board. Unexplained illnesses among Desert Storm Veterans. Arch Intern Med 1995;155:262-268.
[2] U.S. Army Medical Research, Development, Acquisition and Logistics Command (Provisional). The General Well-Being Of Gulf War Era Service Personnel From The States Of Pennsylvania And Hawaii: A Survey. 1994. Office of the Assistant Secretary of Defense - Health Affairs, the Pentagon, Arlington, VA.
[3] Ross MC, Wonders J. An exploration of the characteristics of post-traumatic stress disorder in reserve forces deployed during Desert Storm. Arch Psychiatr Nurs 1993;7:265-269.
[4]Perconte ST, Dietrick AL, Wilson AT, Spiro KJ, Pontius EB. Psychological and war stress symptoms among deployed and non-deployed reservists following the Persian Gulf war. Mil Med 1993;158:516-521.
[5] Sutker PB, Uddo M, Brailey K, Allain An. War-zone trauma and stress-related symptoms in Operation Desert Shield/Storm (ODS) returnees. J Soc Issues 1993;49:33-49.
[6] Report of the Defense Science Board Task Force on Persian Gulf War Health Effects, June 1994. Office of the Under Secretary of Defense for Acquisition and Technology. Washington, DC 20301-3140.
[7]National Institutes of Health Technology Assessment Workshop Panel. The Persian Gulf Experience and Health. JAMA 1994;272:391-395.
[8] Dunn MA, Sidell FR. Progress in medical defense against nerve agents. JAMA 1989;262:649-652.
[9] Sharabi Y, Danon YL, Berkenstadt H, Almog S, Mimouni-Bloch A, Zisman A, Dani S, Atsomon J. Survey of symptoms following intake of pyridostigmine during the Persian Gulf war. Isr J Med Sci 1991;27:656-658.
[10] Cook JE, Wenger CB, Kolka MA. Chronic pyridostigmine bromide administration: Side effects among soldiers working in a Desert Environment. Mil Med 1992;157:250-254.
[11] Keeler JR, Hurst CG, Dunn MA. Pyridostigmine used as a nerve agent pretreatment under wartime conditions. JAMA 1991;266: 693-695.
[12] Brachman PS, Gold H, Plotkins SA, Fekety FR, Werrin M, Ingraham NR. Field evaluation of a human anthrax vaccine. Am J Pub Health 1962;52:632-645.
[13] White CS, Adler WH, McGann VG. Repeated Immunization: Possible adverse effects: Reevaluation of human subjects at 25 years. Ann Intern Med 1974;81:594-600.
[14] Hyams KC, Hanson K, Wigall FS, Escamilla J, Oldfield EC. The impact of infectious diseases on the health of U.S. troops deployed to the Persian Gulf during Operations Desert Shield and Desert Storm. Clin Infect Diseases. 1995;20:1497-1504.
[15] Magill AJ, Grogl M, Gasser RA, Sun W, Oster CN. Visceral infection caused by Leishmani tropica in veterans of Operation Desert Storm. N Engl J Med 1993;328:1383-1387.
[16] Executive Summary, Final Report: Kuwait oil fire health risk assessment, No. 39:26-L192-91, 5 May - 3 December 1991. Department of the Army, U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD 21010-5422.
[17] Report to Congress, United States Gulf Environmental Technical assistance, January 27-July 31, 1991, U.S. Environmental Protection Agency. Gulf Task Force, EPA, Washington, DC 20460.
[18] Korenyi-Both AL, Molnar AC, Korenyi-Both AL, Fidelu-Gort RF. Al Eskan disease: Desert Storm pneumonitis. Mil Med 1992;157:452-462.
[19] United States National Center for Health Statistics. International Classification of Diseases, 9th Revision, Clinical Modification. 1980;Vols 1-3.
[20] Kroenke K, Price R. Symptoms in the community: prevalence, classification, and psychiatric comorbidity. Arch Intern Med 1993;153:2474-2480.
[21] Kroenke K, Arrington ME, Mangeisdorff AD. The prevalence of symptoms in medical outpatients and the adequacy of therapy. Arch Intern Med 1990;160:1685-1689.
[22] Kroenke K, Spitzer RL, Williams JBW, et al. Physical symptoms in primary care: predictors of psychiatric disorders and functional impairment. Arch Fam Med 1994; 3:774-779.
[23] Reidenberg MM, Lowenthal DT: Adverse mondrug reactions. N Engl J Med 1968; 279:678-679
[24] Marple R, Lucey C, Kroenke K, et al. A prospective study of the concerns and expectations in patients presenting with common symptoms; and The 2 week outcome in patients presenting with common physical complaints. Abstracts in Clin Res 41:579A, 1993; and J Gen Intern Med 9 (suppl 2):96, 1994.

[25] Komaroff AL, Buchwald D. Symptoms and signs of Chronic Fatigue Syndrome. Rev Infect Dis 1991;13(suppl 1):S8-S11.
[26] Buchwald D, Garrity D. Comparison of patients with Chronic Fatigue Syndrome, fibromyalgia, and multiple chemical sensitivities. Arch Intern Med 1994;154:2049-2053.
[27] Young MB, Mass AT, Alged JC. A controlled study of primary fibromyalgia syndrome: clinical features and association with other functional syndromes. J Rheumatol 1989;16(suppl 19):62-71.
[28] Mathew RJ, Weinman ML, Mirabi M. Physical symptoms of depression. Br J Psychiatry 1981;139:293-296.
[29] Hudson JI, Goldenberg DL, Pope HG, Keck PE, Schiesinger L. Comorbidity of fibromyalgia with medical and psychiatric disorders. AM J Med 1992;92:363-367.
[30] CDC. Unexplained Illness Among Persian Gulf War Veterans in an Air National Guard Unit: Preliminary Report - 1995. MMWR 1995;44:443-446.
[31] Schappert SM: National Ambulatory Medical Care Survey: 1989 summary. National Center for Health Statistics. Vital Health Stat 1992;13(110).
[32] Spitzer RL, Williams JBW, Kroenke K. Utility of a new procedure for diagnosing psychological conditions in primary care: The PRIME-MD 1000 study. JAMA 1994;272:1749-1756.
[33] Sartorius N, Goldberg D, de Girolamo G, Costa Silva JA, Lecrubier Y, Wittchen HU (eds). Psychological Disorders in General Medical Settings. Hogrete & Huber: Toronto, 1990.
[34] American Psychiatric Association: Diagnostic and Statistical Manual of Psychological Conditions, Fourth Edition. Washington DC, American Psychiatric Association; 1994.
[35] Goldberg D, Huxley P. Common Psychological Conditions: A Bio-Social Approach. Routledge, New York, NY; 1992
[36] Cunningham LS, Kelsey, JL. Epidemiology of musculoskeletal impairments and associated disability. Am J Public Health 1984;74(6):574-9.
[37] McKee K, Gasser RA Jr, Magill AJ, Oster CN. Diagnosis and Treatment of Diseases of Tactical Importance to US CENTCOM Forces 1990. Washington DC: Office of the Surgeon General, U.S. Army, Oct 1990.
[38] Personal communication, Magill AJ, 2 June 95
[39] Price RK, North CS, Wessely S, Fraser VJ. Estimating the prevalence of chronic fatigue syndrome and associated symptoms in the community. Pub Health Reports. 1992; 107:514-22.
[40] Lloyd AR, Hickie I, Boughton CR, Spencer O, Wakefield D. Prevalence of fatigue syndrome in an Australian population. Med Journ Australia. 1990; 153:522-528.
[41] Wolfe F., Smythe HA, Yunus, MB, et al. The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia. Arthr Rheum. 1990;33:160-172.
[42] Gallup Organization. Sleep in America. Princeton, NJ:Author. 1991.
[43] Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle aged adults. N Engl J Med 1993;328:1230-1235.
[44] Bresnitz EA, Goldberg R, Kosinski RM. Epidemiology of obstructive sleep apnea. Epidemiologic Reviews. 1994;16:210227.
[45] Partinen M. Principles and Practice of Sleep Medicine. Philadelphia: W.B. Sanders, 1994.
[46] Morin CM. Insomnia. New York: Guilford Press, 1993;3-15.
[47] Lezak M. Neuropsychological Assessment. 2nd ed. New York: Oxford University Press, 1983.
[48] Miller WR. Psychological deficit in depression. Psychological Bulletin 1975;82:238-260.
[49] Kryger MH, Roth T, Dement W, eds, Principles of sleep medicine. Philadelphia: Sanders, 1989.
[50] Graffman J, Johnson R Jr., Scheffers M Cognitive and mood-state changes in patients with chronic fatigue syndrome. Reviews of Infection Diseases. 1991;13(Supp): 45-52
[51] U.S. Dept of Health & Human Services. Centers for Disease Control. Field Epidemiology Report 95-01. December 19, 1994.
[52] Final Report of Robbins AFB, GA Investigation. Armstrong Laboratory (AL/AOES) Brooks AFB, TX. (TVA-T03704.11-15 April 1994): June 21, 1994.
[53] Rosa C. Spontaneous abortion rate and the Gulf war mobilization. J US Army Med Dept Sep-Oct 1993;P8-8-93-9/10.

[54] Rubin HR, Gandek B, Rogers WH, Kosinski M, McHorney CA, Ware JE. Patients' ratings of outpatient visits in different practice settings: results from the Medical Outcomes Study. JAMA 1993;270:835-40.
[55] Department of Veterans Affairs. Federal activities related to the health of Persian Gulf veterans. Department of Veterans Affairs, March 1995.
[56] Lewis T. The Soldier's Heart and the Effort Syndrome. New York: Paul Hoeber (Ed), 1919:1-103.
[57] DaCosta JM. On irritable heart: A clinical study of a form of functional cardiac disorder and its consequences. Am J Med Sci 1871;61:17-52.
[58] Grinker RR, Spiegel JP. The Syndrome of "Operational Fatigue" (War Neuroses) in returnees. In: Men Under Stress, Maple Press Company, York, PA.
[59] Bogen G. Symptoms in Vietnam Veterans Exposed To Agent Orange. JAMA 1979:242:2391.
[60] Litz BT, Keane TM, Fisher L, Marx B, Monaco V. Physical health complaints in combat-related posttraumatic stress disorder: A preliminary report. J Traumatic Stress 1992; 5:131-141.
[61] Baum A. Toxins, Technology and Natural Disasters. In Vandenbos, G.R. & Bryant, B.K (Ed) Cataclysms, Crises, and Catastrophes: Psychology in Action. Washington, DC: American Psychological Association, 1987.
[62] Fullerton C, Brandt G, Ursano R. Chemical and Biological Weapons: Silent Agents of Terror. In: Emotional aftermath of the Persian Gulf War: Veterans, families, communities and Nations. American Psychiatric Press, (in press/1996).
[63] Pechura CM, Rall D.P.(Eds.): Veterans at Risk: The Health Effects of Mustard Gas and Lewisite. National Academy Press, Washington, D.C., 1993.